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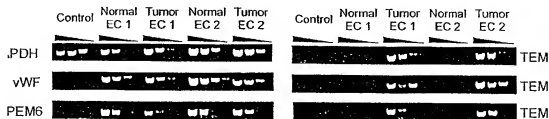
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(54) Title: ENDOTHELIAL CELL EXPRESSION PATTERNS



(57) Abstract: To gain a better understanding of tumor angiogenesis, new techniques for isolating endothelial cells (ECs) and evaluating gene expression patterns were developed. When transcripts from ECs derived from normal and malignant colorectal tissues were compared with transcripts from non-endothelial cells, over 170 genes predominantly expressed in the endothelium were identified. Comparison between normal- and tumor-derived endothelium revealed 79 differentially expressed genes, including 46 that were specifically elevated in tumor-associated endothelium. Experiments with representative genes from this group demonstrated that most were similarly expressed in the endothelium of primary lung, breast, brain, and pancreatic cancers as well as in metastatic lesions of the liver. These results demonstrate that neoplastic and normal endothelium in humans are distinct at the molecular level,

ENDOTHELIAL CELL EXPRESSION PATTERNS

- [01] This application claims the benefit of provisional applications serial number 60/282,850 filed April 11, 2001, and 60,308,829 filed August 1, 2001, the disclosures of which are expressly incorporated herein.
- [02] The U.S. government retains certain rights in the invention by virtue of the provisions of National Institutes of Health grants CA57345 and CA43460, which supported this work.

TECHNICAL FIELD OF THE INVENTION

- [03] This invention is related to the area of angiogenesis and anti-angiogenesis. In particular, it relates to genes which are characteristically expressed in tumor endothelial and normal endothelial cells.

BACKGROUND OF THE INVENTION

- [04] It is now widely recognized that tumors require a blood supply for expansive growth. This recognition has stimulated a profusion of research on tumor angiogenesis, based on the idea that the vasculature in tumors represents a potential therapeutic target. However, several basic questions about tumor endothelium remain unanswered. For example, are vessels of tumors qualitatively different from normal vessels of the same tissue? What is the relationship of tumor endothelium to endothelium of healing wounds or other physiological or pathological forms of angiogenesis? The answers to these questions critically impact on the potential for new therapeutic approaches to inhibit angiogenesis in a specific manner.

- [05] There is a continuing need in the art to characterize the vasculature of tumors relative to normal vasculature so that any differences can be exploited for therapeutic and diagnostic benefits.
- [06] One technique which can be used to characterize gene expression, or more precisely gene transcription, is termed serial analysis of gene expression (SAGE). Briefly, the SAGE approach is a method for the rapid quantitative and qualitative analysis of mRNA transcripts based upon the isolation and analysis of short defined sequence tags (SAGE Tags) corresponding to expressed genes. Each Tag is a short nucleotide sequences (9-17 base pairs in length) from a defined position in the transcript. In the SAGE method, the Tags are dimerized to reduce bias inherent in cloning or amplification reactions. (See, US Patent 5,695,937) SAGE is particularly suited to the characterization of genes associated with vasculature stimulation or inhibition because it is capable of detecting rare sequences, evaluating large numbers of sequences at one time, and to provide a basis for the identification of previously unknown genes.

SUMMARY OF THE INVENTION

- [07] One embodiment of the invention provides an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 17, 19, and 44, as shown in SEQ ID NO: 196, 200, 212, 230, 232, and 271, respectively. The molecule can be, for example, an intact antibody molecule, a single chain variable region (ScFv), a monoclonal antibody, a humanized antibody, or a human antibody. The molecule can optionally be bound to a cytotoxic moiety, bound to a therapeutic moiety, bound to a detectable moiety, or bound to an anti-tumor agent.

- [08] According to another embodiment of the invention a method of inhibiting neoangiogenesis is provided. An effective amount of an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 17, 19, 22, and 44, as shown in SEQ ID NO: 196, 200, 212, 230, 232, 238, and 271, respectively, is administered to a subject in need thereof. Neoangiogenesis is consequently inhibited. The subject may bear a vascularized tumor, may have polycystic kidney disease, may have diabetic retinopathy, may have rheumatoid arthritis, may have psoriasis, for example.
- [09] Another aspect of the invention is a method of inhibiting tumor growth. An effective amount of an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 17, 19, 22, and 44, as shown in SEQ ID NO: 196, 200, 212, 230, 232, 238, and 271, respectively, is administered to a human subject bearing a tumor. The growth of the tumor is consequently inhibited.
- [10] Still another aspect of the invention provides an isolated molecule comprising an antibody variable region which specifically binds to a TEM protein selected from the group consisting of: 3, 9, 17, 19, and 44, as shown in SEQ ID NO: 200, 212, 230, 232, and 271, respectively. The molecule can be, for example, an intact antibody molecule, a single chain variable region (ScFv), a monoclonal antibody, a humanized antibody, or a human antibody. The molecule can optionally be bound to a cytotoxic moiety, bound to a therapeutic moiety, bound to a detectable moiety, or bound to an anti-tumor agent.
- [11] According to still another aspect of the invention an isolated and purified human transmembrane protein is provided. The protein is selected from the group consisting of: TEM 3, 9, 17, and 19 as shown in SEQ ID NO: 200, 212, 230, and 232, respectively.

- [12] Yet another aspect of the invention is an isolated and purified nucleic acid molecule comprising a coding sequence for a transmembrane TEM selected from the group consisting of: TEM 3, 9, 17, and 19 as shown in SEQ ID NO: 200, 212, 230, and 232, respectively. The isolated and purified nucleic acid molecule may optionally comprise a coding sequence selected from those shown in SEQ ID NO: 199, 211, 229, and 231.
- [13] Still another aspect of the invention is a recombinant host cell which comprises a nucleic acid molecule. The nucleic acid molecule comprises a coding sequence for a transmembrane TEM selected from the group consisting of: TEM 3, 9, 17, and 19 as shown in SEQ ID NO: 200, 212, 230, and 232, respectively. The recombinant host cell optionally comprises a coding sequence selected from those shown in SEQ ID NO: 199, 211, 229, and 231.
- [14] According to one embodiment of the invention a method is provided for inducing an immune response in a mammal. A nucleic acid molecule comprising a coding sequence for a human transmembrane protein selected from the group consisting of: TEM 1, 3, 9, 13, 17, 19, 22, 30, and 44 as shown in SEQ ID NO: , respectively, is administered to the mammal. An immune response to the human transmembrane protein is thereby induced in the mammal. Optionally the coding sequence is shown in SEQ ID NO: 196, 200, 212, 220, 230, 232, 238, 250 and 271.
- [15] According to yet another embodiment of the invention a method of inducing an immune response in a mammal is provided. A purified human transmembrane protein selected from the group consisting of: TEM 1, 3, 9, 13, 17, 19, 22, 30, and 44 as shown in SEQ ID NO: 196, 200, 212, 220, 230, 232, 238, 250 and 271, respectively, is administered to the mammal. An immune response to the human transmembrane protein is thereby induced in the mammal.

- [16] Another aspect of the invention is a method for identification of a ligand involved in endothelial cell regulation. A test compound is contacted with an isolated and purified human transmembrane protein selected from the group consisting of 1, 3, 9, 13, 17, 30, 19, and 44 as shown in SEQ ID NO: 196, 200, 212, 220, 230, 232, 250, and 271. The isolated and purified human transmembrane protein is also contacted with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 13, 17, 30, 19, and 44 as shown in SEQ ID NO: 196, 200, 212, 220, 230, 232, 250, and 271 respectively. Binding of the molecule comprising an antibody variable region to the human transmembrane protein is determined. A test compound which diminishes the binding of the molecule comprising an antibody variable region to the human transmembrane protein is identified as a ligand involved in endothelial cell regulation.
- [17] Yet another aspect of the invention is a method for identification of a ligand involved in endothelial cell regulation. A test compound is contacted with a cell comprising a human transmembrane protein selected from the group consisting of 1, 3, 9, 17, and 19 as shown in SEQ ID NO: 196, 200, 212, 230, and 232. The cell is also contacted with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 17, and 19 as shown in SEQ ID NO: 196, 200, 212, 230, and 232, respectively. Binding of the molecule comprising an antibody variable region to the cell is determined. A test compound which diminishes the binding of the molecule comprising an antibody variable region to the cell is identified as a ligand involved in endothelial cell regulation.
- [18] Yet another aspect of the invention is a method for identification of a ligand involved in endothelial cell regulation. A test compound is contacted with a human transmembrane protein selected from the group consisting of 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29,

40, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 196, 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275. Binding of a test compound to the human transmembrane protein is determined. A test compound which binds to the protein is identified as a ligand involved in endothelial cell regulation.

- [19] Another embodiment of the present invention is a soluble form of a human transmembrane protein selected from the group consisting of: TEM 1, 3, 9, 17, 19, 22, 30, and 44 as shown in SEQ ID NO: 196, 200, 212, 230, 232, 238, 250, and 271 respectively. The soluble forms lack transmembrane domains. The soluble form may consist of an extracellular domain of the human transmembrane protein.
- [20] Also provided by the present invention is a method of inhibiting neoangiogenesis in a patient. A soluble form of a human transmembrane protein is administered to the patient. Neoangiogenesis in the patient is consequently inhibited. The patient may bear a vascularized tumor, may have polycystic kidney disease, may have diabetic retinopathy, may have rheumatoid arthritis, or may have psoriasis, for example.
- [21] Another embodiment of the invention provides a method of inhibiting neoangiogenesis in a patient. A soluble form of a human transmembrane protein is administered to the patient. Neoangiogenesis in the patient is consequently inhibited. The patient may bear a vascularized tumor, may have polycystic kidney disease, may have diabetic retinopathy, may have rheumatoid arthritis, or may have psoriasis, for example.
- [22] According to still another aspect of the invention a method of identifying regions of neoangiogenesis in a patient is provided. A molecule comprising an antibody variable region which specifically binds to an extracellular

domain of a TEM protein selected from the group consisting of: 1, 3, 9, 13, 17, 19, 22, 30, and 44, as shown in SEQ ID NO: 196, 200, 212, 220, 230, 232, 238, 250, and 271, respectively, is administered to a patient. The molecule is bound to a detectable moiety. The detectable moiety is detected in the patient, thereby identifying neoangiogenesis.

- [23] According to another aspect of the invention a method is provided for inducing an immune response to tumor endothelial cells in a patient. A mouse TEM protein selected from the group consisting of: 1, 2, 3, 9, 13, 17, 19, 22, and 30 as shown in SEQ ID NO: 291, 293, 299, 295, 303, 297, 301, 305, and 307, is administered to a patient in need thereof. An immune response to a human TEM protein is consequently induced.
- [24] Still another embodiment of the invention is a method of screening for neoangiogenesis in a patient. A body fluid collected from the patient is contacted with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 1, 3, 9, 17, 19, and 44, as shown in SEQ ID NO: 196, 200, 212, 230, 232, and 271, respectively. Detection of cross-reactive material in the body fluid with the molecule indicates neo-angiogenesis in the patient.
- [25] Still another embodiment of the invention provides a method of inhibiting neoangiogenesis in a patient. A molecule comprising an antibody variable region which specifically binds to a TEM protein selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40 as shown in SEQ ID NO: 202, 206, 208, 214, 218, 223 and 224, 234, 242, 244, 252, 257, 259, 261, 263, and 265, is administered to the patient. Neoangiogenesis in the patient consequently inhibited.
- [26] Yet another aspect of the invention is a method of screening for neoangiogenesis in a patient. A body fluid collected from the patient is

contacted with a molecule comprising an antibody variable region which specifically binds to a TEM protein selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40, as shown in SEQ ID NO: 202, 206, 208, 214, 218, 223 & 224, 234, 242, 244, 252, 257, 259, 261, 263, and 265, respectively. Detection of cross-reactive material in the body fluid with the molecule indicates neoangiogenesis in the patient.

- [27] Also provided by the present invention is a method of promoting neoangiogenesis in a patient. A TEM protein selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40, as shown in SEQ ID NO: 202, 206, 208, 214, 218, 223 & 224, 234, 242, 244, 252, 257, 259, 261, 263, and 265, is administered to a patient in need of neoangiogenesis. Neoangiogenesis in the patient is consequently stimulated.
- [28] One embodiment of the invention provides a method of promoting neoangiogenesis in a patient. A nucleic acid molecule encoding a TEM protein selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40, as shown in SEQ ID NO: 201, 205, 207, 213, 217, 221 & 222, 233, 241, 243, 251, 256, 258, 260, 262, and 264, is administered to a patient in need of neoangiogenesis. The TEM protein is consequently expressed and neoangiogenesis in the patient is stimulated.
- [29] Another embodiment of the invention provides a method of screening for neoangiogenesis in a patient. A TEM protein selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40, as shown in SEQ ID NO: 202, 206, 208, 214, 218, 223 & 224, 234, 242, 244, 252, 257, 259, 261, 263, and 265, respectively, is detected in a body fluid collected from the patient. Detection of the TEM protein indicates neoangiogenesis in the patient.
- [30] Another aspect of the invention is a method of screening for neoangiogenesis in a patient. A nucleic acid encoding a TEM protein

selected from the group consisting of: 4, 6, 7, 10, 12, 14, 20, 25, 27, 31, 36, 37, 38, 39, and 40 is detected in a body fluid collected from the patient. The nucleic acid is selected from the group consisting of those shown in SEQ ID NO: 201, 205, 207, 213, 217, 221 & 222, 233, 241, 243, 251, 256, 258, 260, 262, and 264. Detection of the TEM protein indicates neoangiogenesis in the patient.

- [31] Yet another embodiment of the invention is an isolated and purified nucleic acid molecule which encodes a NEM protein selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289. The nucleic acid molecule optionally comprises a coding sequence as shown in SEQ ID NO: 278, 282, 284, and 288. The nucleic acid may be maintained in a recombinant host cell.
- [32] The present invention also provides an isolated and purified NEM protein selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289.
- [33] The present invention further provides an isolated molecule comprising an antibody variable region which specifically binds to a NEM protein selected from the group consisting of: 14, 22, 23, and 33, as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289.
- [34] An additional embodiment of the present invention is a method of inhibiting neoangiogenesis. An effective amount of a NEM protein selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289 is administered to a subject in need thereof. Neoangiogenesis is thereby inhibited.
- [35] A still further embodiment of the invention is a method to identify candidate drugs for treating tumors. Cells which express one or more TEM genes selected from the group consisting of: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14,

15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 40, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: : 195, 197, 199, 201, 203, 205, 207, 209, 211, 213, 215, 217, 221 & 222, 225, 227, 229, 231, 233, 235, 237, 239, 241, 243, 245, 247, 249, 251, 253, 255, 256, 258, 260, 262, 266, 268, 270, 272, and 274, respectively, are contacted with a test compound. Expression of said one or more TEM genes is determined by hybridization of mRNA of said cells to a nucleic acid probe which is complementary to said mRNA. A test compound is identified as a candidate drug for treating tumors if it decreases expression of said one or more TEM genes. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs. Test compounds which increase expression can be identified as candidates for promoting wound healing.

- [36] Yet another embodiment of the invention is a method to identify candidate drugs for treating tumors. Cells which express one or more TEM proteins selected from the group consisting of: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 40, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 196, 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275, respectively, are contacted with a test compound. The amount of said one or more TEM proteins in said cells is determined. A test compound is identified as a candidate drug for treating tumors if it decreases the amount of one or more TEM proteins in said cells. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs. Alternatively, a test compound which increases the amount of one or more TEM proteins in said cells is identified as a candidate drug for treating wound healing.

- [37] According to another aspect of the invention a method is provided to identify candidate drugs for treating tumors. Cells which express one or more TEM proteins selected from the group consisting of: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 40, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 196, 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275, respectively, are contacted with a test compound. Activity of said one or more TEM proteins in said cells is determined. A test compound is identified as a candidate drug for treating tumors if it decreases the activity of one more TEM proteins in said cells. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs. Optionally the cells are endothelial cells. If a test compound increases the activity of one more TEM proteins in said cells it can be identified as a candidate drug for treating wound healing.
- [38] An additional aspect of the invention is a method to identify candidate drugs for treating patients bearing tumors. A test compound is contacted with recombinant host cells which are transfected with an expression construct which encodes one or more TEM proteins selected from the group consisting of 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 40, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275, respectively. Proliferation of said cells is determined. A test compound which inhibits proliferation of said cells is identified as a candidate drug for treating patients bearing tumors. A test compound which stimulates proliferation of said

cells is identified as a candidate drug for promoting neoangiogenesis, such as for use in wound healing.

- [39] Another embodiment of the invention provides a method to identify candidate drugs for treating tumors. Cells which express one or more NEM genes selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 278, 282, 284, and 288, respectively, are contacted with a test compound. Expression of said one or more NEM genes is determined by hybridization of mRNA of said cells to a nucleic acid probe which is complementary to said mRNA. A test compound is identified as a candidate drug for treating tumors if it increases expression of said one or more NEM genes. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more NEMs.
- [40] According to another aspect of the invention a method is provided to identify candidate drugs for treating tumors. Cells which express one or more NEM proteins selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289, are contacted with a test compound. The amount of said one or more NEM proteins in said cells is determined. A test compound is identified as a candidate drug for treating tumors if it increases the amount of one more NEM proteins in said cells. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more NEMs.
- [41] An additional aspect of the invention is a method to identify candidate drugs for treating tumors. Cells which express one or more NEM proteins selected from the group consisting of: 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289, are contacted with a test compound. Activity of said one or more NEM proteins in said cells is determined. A test compound is identified as a candidate drug for treating tumors if it

increases the activity of said one or more NEM proteins in said cells. Optionally the cells are endothelial cells. Alternatively or additionally, the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more NEMs.

- [42] Still another embodiment of the invention provides a method to identify candidate drugs for treating patients bearing tumors. A test compound is contacted with recombinant host cells which are transfected with an expression construct which encodes one or more NEM proteins selected from the group consisting of 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289. Proliferation of said cells is determined. A test compound which stimulates proliferation of said cells is identified as a candidate drug for treating patients bearing tumors.
- [43] Another aspect of the invention is a method for identifying endothelial cells. One or more antibodies which bind specifically to a TEM or NEM protein selected from the group consisting of TEM: 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 30, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275 and NEM 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289, is contacted with a population of cells. Cells in the population which have bound to said antibodies are detected. Cells which are bound to said antibodies are identified as endothelial cells. Optionally cells which have bound to said antibodies are isolated from cells which have not bound.
- [44] Still another aspect of the invention is a method for identifying endothelial cells. One or more nucleic acid hybridization probes which are complementary to a TEM or NEM gene nucleic acid sequence selected from the group consisting of TEM: 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16,

17, 19, 20, 21, 22, 24, 25, 27, 28, 29, 30, 31, 33, 35, 36, 37, 38, 39, 41, 42, 44, 45, and 46 as shown in SEQ ID NO: 198, 200, 202, 204, 206, 208, 210, 212, 214, 216, 218, 223 & 224, 226, 228, 230, 232, 234, 236, 238, 240, 242, 244, 246, 248, 250, 252, 254, 358, 257, 259, 261, 263, 267, 269, 271, 273, and 275 and NEM 14, 22, 23, and 33 as shown in SEQ ID NO: 279, 283, 285, 286, 287, and 289, is contacted with nucleic acids of a population of cells. Nucleic acids which have specifically hybridized to said nucleic acid hybridization probes are detected. Cells whose nucleic acids specifically hybridized are identified as endothelial cells.

[45] Yet another embodiment of the invention is a method of inhibiting neoangiogenesis. An effective amount of an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of a mouse TEM protein selected from the group consisting of: 1, 2, 3, 9, 17, and 19, as shown in SEQ ID NO: 291, 293, 299, 295, 297, and 301, respectively, is administered to a subject in need thereof. Neoangiogenesis is thereby inhibited. The subject may be a mouse, may bear a vascularized tumor, may have polycystic kidney disease, may have diabetic retinopathy, may have rheumatoid arthritis, or may have psoriasis, for example.

[46] These and other embodiments which will be apparent to those of skill in the art upon reading the specification provide the art with reagents and methods for detection, diagnosis, therapy, and drug screening pertaining to neoangiogenesis and pathological processes involving or requiring neoangiogenesis.

BRIEF DESCRIPTION OF THE DRAWINGS

[47] Fig. 1A-1B. vWF expression in colorectal cancers. vWF (red stain) was detected in vessels by in situ hybridization. At low power magnification (Fig. 1A) vessels were often surrounded by a perivascular cuff of viable cells

(red arrows), with a ring of necrotic cells evident at the periphery (black arrows). At high power magnification (Fig. 1.B) the expression of vWF (red) was clearly localized to the vessels. Sections were counterstained with methyl green.

- [48] Fig. 2A-2D. Purification of Endothelial Cells (ECs) from human normal and malignant tissue. (Fig. 2A) Vessels (red) of frozen sections were stained by immunofluorescence with the P1H12 monoclonal antibody (Chemicon, Temecula, CA) and detected using a biotinylated goat anti-mouse IgG secondary antibody followed by rhodamine-linked streptavidin. The region stained is from within the lamina propria of normal colonic mucosa. Note that the larger vessels (arrowheads) and capillaries (arrows) are positive, and staining of hematopoietic cells was undetectable. E-cadherin positive epithelial cells (green) at the edge of the crypt were simultaneously visualized using a rabbit polyclonal antibody (Santa Cruz, Santa Cruz, CA), followed by a goat anti-rabbit IgG secondary antibody labelled with alexa (Molecular Probes, Eugene, OR). Sections were imaged at 60X magnification using confocal microscopy. (Fig. 2.B) To isolate pure populations from collagenase dispersed tissues, the epithelial and hematopoietic cell fractions were sequentially removed by negative selection with magnetic beads. The remaining cells were stained with P1H12 and ECs were isolated by positive selection with magnetic beads. (Fig. 2.C) RT-PCR analysis used to assess the purity of the EC preparations. Semiquantitative PCR analysis was performed on cDNA generated either directly from colorectal cancer tissue (unfractionated tumor) or from purified ECs isolated from normal colonic mucosa (normal EC fraction) or colorectal cancer (tumor EC fraction). PCR amplification of the epithelial specific marker cytokeratin 20 (CK20), demonstrated its expression was limited to the unfractionated tumor. Two endothelial specific markers, vWF and VE-cadherin (VE-Cad) showed robust amplification only in the endothelial fractions, validating the purity and enrichment protocol shown in (Fig. 2.B). The ubiquitous housekeeping enzyme GAPDH was observed in all samples.

No signal was detected in the no-template (NT) control. cDNA templates were diluted 1:10, 1:100, 1:1000, 1:4000, and 1:40,000 as indicated by the declining wedge. (Fig. 2.D) The relative expression level of select genes was determined by measuring the tag abundance from several SAGE libraries combined into four groups. The first was composed of ~193,000 tags from the two in vivo-derived EC preparations (Endothelial Cell Fraction) while the second contained a single library of ~57,000 tags containing macrophages and other leukocytes derived from the negative selection (Hematopoietic Fraction). The fourth library contained ~401,000 tags from cultured HUVEC and HMVEC (Endothelial Cells in Culture), and the fourth consisted of ~748,000 tags from 6 colon cancer cell lines in culture (Epithelial Cells). After normalization, the library with the highest tag number for each marker was given a value of 100%, and the corresponding relative expression levels of the remaining 3 libraries was plotted on the ordinate. Note the high level of CD31 present on hematopoietic cells, the likely cause of the impurity of the initial endothelial selection, compared with the selectivity of PIH12.

[49] Fig. 3A- 3E. Expression of Pan-Endothelial Markers (PEMs) is limited to ECs. The endothelial origin of PEMs identified by SAGE was confirmed using a highly sensitive in situ hybridization assay. Localization of novel PEMs to the ECs was demonstrated by examining two representative PEMs, PEM3 (Fig. 3A) and PEM6 (Fig. 3B) in lung cancer and colon cancer, respectively. Hevin expression was readily detected in the ECs of a colon tumor (Fig. 3C) despite its low level of expression in cultured ECs. Expression of VEGFR2 was readily detectable in the ECs of both normal (Fig. 3D) and malignant colon tissue (Fig. 3E).

[50] Fig. 4A-4J. Expression of Tumor Endothelial Markers (TEMs). (Fig. 4A) RT-PCR analysis confirmed the tumor specific expression of selected novel TEMs. Semiquantitative PCR analysis was performed on cDNA generated either from purified epithelial cells as a negative control (Control) or from purified ECs isolated from normal colonic mucosa (Normal ECs) or

colorectal cancer (Tumor ECs) from two different patients. Two endothelial specific markers, vWF and PEM6 showed robust amplification only in the endothelial fractions whereas the ubiquitous housekeeping enzyme GAPDH was observed in all samples. TEM1 (BSC-TEM1), TEM 17 (BSC-TEM7) and TEM22 (BSC-TEM9) were specifically expressed in tumor compared to normal ECs. The cDNA template was diluted 1:10, 1:100, 1:1000, and 1:10,000 as indicated by the declining wedge. (Fig. 4 B- 4J) The endothelial origin of TEMs identified by SAGE was confirmed using in situ hybridization as in Fig 3. Expression of TEM 1 (BSC-TEM1) (Fig. 4 B) and TEM17 (BSC-TEM7) (Fig. 4 C) was demonstrated to be highly specific to the ECs in colorectal cancers; sections were imaged in the absence of a counterstain to show the complete lack of detectable expression in the non-endothelial cells of the tumor. Expression of TEM17 (BSC-TEM7) in ECs was demonstrated in a metastatic liver lesion from a primary colorectal cancer (Fig. 4 D), a lung (Fig. 4 E), breast (Fig. 4 F), pancreatic (Fig. 4 G) and brain cancer (Fig. 4 H), as well as in a sarcoma (Fig. 4 I). TEM 17 (BSC-TEM7) was also localized to vessels during normal physiological angiogenesis of the corpus luteum (Fig. 4 J).

DETAILED DESCRIPTION OF THE INVENTION

[51] We identified 46 human genes that were expressed at significantly higher levels (> 10 -fold) in tumor endothelium than in normal endothelium, and 33 genes that were expressed at significantly lower levels in human tumor versus normal endothelium. See Tables 2 and 4, respectively. Most of these genes were either not expressed or expressed at relatively low levels in Endothelial Cells (ECs) maintained in culture. Moreover, we identified 93 genes which are expressed in both normal and tumor human endothelium. Interestingly, the tumor endothelium genes were expressed in all tumors tested, regardless of its tissue or organ source. Most tumor endothelium genes were also expressed in corpus luteum and wounds.

[52] As the work has progressed, we have refined and classified our original 46 tumor endothelial markers. We have named these markers TEMs and renumbered them consecutively by the prevalence of their tags in our SAGE analysis. Originally we had not used a consecutive numbering system. Our non-consecutive numbering system has been renamed as BSC-TEMs. For most of the original 46 SAGE Tags, we now provide full-length nucleic acid and protein sequence. In some cases, the sequences were obtained through the public databases, in others the sequences were obtained by cloning and through the use of gene prediction tools. In some cases, we found SAGE Tags corresponding to genes having different splice variants or with known polymorphisms. For example, in one case the SAGE Tag BSC-TEM3 has been found to hybridize to an alternatively spliced form of the transcript encoding BSC-TEM7. The proteins encoded by the two transcripts are the same; therefore they are cumulatively called TEM7. A highly related sequence was found via homology searches, BSC-TEM7R. This paralog sequence is now called TEM3. See Table 2, which follows, showing tumor endothelial markers by order of prevalence (except for TEM 3). Column 1 indicates the prevalence number. Column 2 indicates the original nomenclature. Column 3 indicates the short tags. Column 4 indicates the long tags. Column 5 indicates the accession number in GenBank. Column 6 indicates the sequence identifiers for the short tag, the long tag, the full nucleic acid, and the protein. Column 7 provides a functional description, which is expanded below in the text.

TEM1	BSC-TEM1	GGGGCTGCC CA	GGGGCTGCCCAGCT GA	NM020404	SEQ ID NO : 94, 309, 195, 196	tumor endothelial marker 1 precursor
TEM 2	BSC-TEM2	GATCTCCGT GT			SEQ ID NO: 95, 197, 198	sapiens tumor endothelial marker 2 (BSC-TEM2) mRNA/mouse Ras, dexamethasone-induced 1 (RASD1), mRNA
TEM 3	BSC-TEM7 R				SEQ ID NO: 199, 200, 359	human ortholog of mouse paralog of mouse TEM-7
TEM 4		CTTCTTTGA G	CTTCTTTGAGTTTT AA	AB034203	SEQ ID NO: 97, 311, 201, 202	Homo sapiens dickkopf-3 (DKK-3) mRNA,
TEM 5	BSC-TEM4 C	TATTAAGTCT C	TATTAAGTCTCTTTG GA		SEQ ID NO: 98, 312, 203, 204	Tumor endothelial marker 4
TEM 6		CAGGAGACC CC	CAGGAGACCCACAGG CCC	X57766	SEQ ID NO: 99, 314, 205, 206	Human stromelysin-3 mRNA.
TEM 7		GGAAATGTC AA	GGAAATGTCAGCAA GTA	BC002576	SEQ ID NO: 100, 315, 207, 208	matrix metalloproteinase 2 (gelatinase A, 72kD gelatinase, 72kD type IV collagenase)

TEM 8	CCTGGTTCA GT			SEQ ID NO:101, 316, 209, 210	HeyL transcription factor
TEM 9 BSC- TEM5	TTTTAAGAA C	TTTTAAGAACTCGG GT		SEQ ID NO:102, 317, 211, 212	
TEM 10	TTTGGTTTC C	TTTGGTTTCCAAAA GA	J03464, M18057, X02488	SEQ ID NO:103, 319, 213, 214	Human collagen alpha-2 type I mRNA, complete cds, clone pHCOL2A1.
TEM 11	ATTTTGTATG A	ATTTTGTATGATTTT TA	NM_00250 8	SEQ ID NO:104, 321, 215, 216	nldogen/entactin
TEM 12	ACTTTAGATG G	ACTTTAGATGGGAA GCC	X52022	SEQ ID NO:105, 322, 217, 218	H.sapiens RNA for type VI collagen alpha3 chain.
TEM 13	GAGTGAGAC CC	GAGTGAGACCCAGG AGC	IM11749	SEQ ID NO:106, 324, 219, 220	Human Thy-1 glycoprotein gene, complete cds.
TEM 14	GTACACACA CC	GTACACACACCCCC ACC		SEQ ID NO:107, 325, 221, 223	Cystatin SN

TEM 14	GTACACACA CC	GTACACACACCCCC ACC	X54687	SEQ ID NO:107, 325, 222, 224	H.sapiens mRNA for cystatin S.
TEM 15	CCACAGGG AT	CCACAGGGGATTCT CCT	NM_00009 0	SEQ ID NO:108, 327, 225, 226	
TEM BSC- 16 TEM6	TTAAAAGTCA C	TTAAAAGTCACTGTG CA		SEQ ID NO:109, 328, 227, 228	
TEM BSC- 17 TEM7	ACAGACTGTT A	ACAGACTGTTAGCC AAG	AF279144	SEQ ID NO:110, 329, 229, 230	Human Tumor endothelial marker 7
TEM 18	CCACTGCAA CC			SEQ ID NO:111	
TEM BSC- 19 TEM8	CTATAGGAG AC			SEQ ID NO:112, 330, 231, 232	
TEM 20	GTTCCACAG AA		NM_00008 9	SEQ ID NO:113, 233, 234	collagen, type I, alpha 2 (COL1A2)

TEM 21	TACCACCTC CC	TACCACCTCCCTTTC CT	SEQ ID NO:114, 331, 235, 236	Homo sapiens mRNA; cDNA DKFZp762B245 (from clone DKFZp762B245);
TEM 22	BSC- TEM9 T	GCCCTTTCTC GTT	SEQ ID NM_00603 9 SEQ ID NO:115, 334, 237, 238	endocytic receptor (macrophage mannose receptor family) (KIAA0709),
TEM 23	TTAAATAGCA C	TTAAATAGCACCTTT AG	SEQ ID NO:116, 335	no match
TEM 24	AGACATACT GA	AGACATACTGACAG AAT	SEQ ID NM_02284 8 SEQ ID NO:117, 336, 239, 240	Homo sapiens mRNA; cDNA DKFZp434G162 (from clone DKFZp434G162);
TEM 25	TCCCCCAGG AG	TCCCCCAGGAGCCA CCG	SEQ ID L35279, NM_00612 9 SEQ ID NO:118, 338, 241, 242	Homo sapiens (clone KT2) bone morphogenetic protein-1 (BMP-1) mRNA
TEM 26	AGCCCAAAG TG		SEQ ID NO:119	No Match
TEM 27	ACTACCATAA C		SEQ ID NM_00306 2 SEQ ID NO:120, 243, 244	Homo sapiens mRNA for MEGF5, partial cds.
TEM 28	TACAAATCGT T	TACAAATCGTTGTCA AA	SEQ ID NM_01485 9 SEQ ID NO:121, 339, 245, 246	Homo sapiens mRNA for KIAA0672 protein, complete cds.

TEM 29	TTGGGTGAA AA			SEQ ID NO:122, 247, 248	ESTs (2 unigene clusters)
TEM 30	CATTATCCAA A	CATTATCCAAAAACA AT	THC53402 9, X68742, AI262158, AI88747, AI394565, AA679721	SEQ ID NO:123, 340, 249, 250	integrin, alpha 1
TEM 31	AGAAACCAC GG	AGAAACCACGGAAA TGG	NM_00184 5	SEQ ID NO:124, 341, 251, 252	hypothetical protein KIAA1164
TEM 32	ACCAAAACC AC			SEQ ID NO:125	no match
TEM 33	TGAAATAAAC		NM_00025 5	SEQ ID NO:126, 253, 254	methylmalonyl Coenzyme A mutase
TEM 34	TTTGTTTCC			SEQ ID NO:127	no match
TEM 35	GTGGAGACG GA	GTGGAGACGGACTC TGT	ESTAI186 535	SEQ ID NO:128, 345, 255, 358	est

TEM 36	TTTGTGTTGT A	TTTGTGTTGTATATT TA	NM_00437 0	SEQ ID NO:129, 346, 256, 257	est
TEM 37	TTATGTTTAA T	TTATGTTTAAATAGTT GA	NM_00234 5	SEQ ID NO:130, 347, 258, 259	Human lumican mRNA, complete cds.
TEM 38	TGGAATGA C	TGGAATGACCCAA AAA	NM_00008 8	SEQ ID NO:131, 348, 260, 261	collagen type1 alpha1
TEM 39	TGCCACACA GT	TGCCACACAGTGAC TTG	NM_00323 9	SEQ ID NO:132, 350, 262, 263	Human transforming growth factor-beta 3 (TGF-beta3) mRNA, complete
TEM 40	GATGAGGAG AC	GATGAGGAGACTGG CAA	SEQ ID NO:133, 351, 264, 265	collagen, type I, alpha 2	
TEM 41	ATCAAAGGTT T	ATCAAAGGTTTGATT TA	SEQ ID NO:134, 352, 266, 267	est	
TEM 42	AGTCACTAGT	AGTCACTAGTACAT AA	NM_02522 6	SEQ ID NO: 135, 353, 268, 269	ESTs

TEM 43	TTCGGTTGG TC	TTCGGTTGGTCAAA GAT		SEQ ID NO:136, 354	No match
TEM 44	CCGACACG GG	CCCCACACGGGCAA GCA	NM_01835 4v	SEQ ID NO: 137, 355, 270, 271	Homo sapiens cDNA FLJ11190 fis, clone PLACE1007583.
TEM 45	GGCTTGCCT TT	GGCTTGCCTTTTGT AT	NM_00036 6	SEQ ID NO:138, 356, 272, 273	est
TEM 46	ATCCCTTCCC G	ATCCCTTCCCGCCA CAC	NM_00268 8	SEQ ID NO:139, 357, 274, 275	Homo sapiens mRNA for peanut-like protein 1, PNUTL1 (hCDCrel-1).

[53] The studies described below provide the first definitive molecular characterization of ECs in an unbiased and general manner. They lead to several important conclusions that have direct bearing on long-standing hypotheses about angiogenesis. First, it is clear that normal and tumor endothelium are highly related, sharing many endothelial cell specific markers. Second, it is equally clear that the endothelium derived from tumors is qualitatively different from that derived from normal tissues of the same type and is also different from primary endothelial cultures. Third, these genes are characteristically expressed in tumors derived from several different tissue types, documenting that tumor endothelium, in general, is different from normal endothelium. Fourth, the genes expressed differentially in tumor endothelium are also expressed during other angiogenic processes such as corpus luteum formation and wound healing. It is therefore more appropriate to regard the formation of new vessels in tumors as "neoangiogenesis" rather than "tumor angiogenesis" *per se*. This distinction is important from a variety of perspectives, and is consistent with the idea that tumors recruit vasculature using much of, or basically the same signals elaborated during other physiologic or pathological processes. That tumors represent "unhealed wounds" is one of the oldest ideas in cancer biology.

[54] The nature and precise biological function of many of the Tumor Endothelial Markers (TEMs) identified here are unknown. Of the previously characterized genes shown in Table 2, it is intriguing that several encode proteins involved in extracellular matrix formation or remodelling (TEM 6, TEM 6, TEM 10, TEM 7, TEM 11, TEM 12, TEM 14, TEM 20, TEM 24, TEM 25, TEM 27, TEM 37, TEM 38, and TEM 40.) Deposition of extracellular matrix is likely critical to the growth of new vessels. Finally, it is perhaps not surprising that so many of the endothelial-specific transcripts identified here, whether expressed only in neovasculature or in endothelium in general, have not been previously characterized, and some are not even represented in EST databases. In part, this may be due to the fact that the EST databases are heavily biased toward certain tissues, but moreover, may be due to the fact that even in highly vascularized tissues endothelial cells are still a

relatively small proportion of the population. Thus, the sensitivity of the SAGE method is a particularly appropriate tool.

- [55] Sequence and literature study has permitted the following identifications to be made among the family of TEM proteins. TEM proteins have been identified which contain transmembrane regions. These include TEM 1, TEM 3, TEM 9, TEM 13, TEM 17, TEM 19, TEM 22, TEM 30, and TEM 44. TEM proteins have been identified which are secreted proteins, including TEM 4, TEM 6, TEM 7, TEM 10, TEM 12, TEM 14, TEM 20, TEM 25, TEM 27, TEM 31, TEM 36, TEM 37, TEM 38, and TEM 39. HeyL (TEM 8) is a transcription factor which may be involved in regulating TEMs as one or more groups. The protein corresponding to the tag for TEM44 was found in the public databases, but no biological function has yet been ascribed to it.
- [56] TEM 1 has been named endosialin in the literature. It has a signal sequence at amino acids 1-17 and a transmembrane domain at amino acids 686-708. Thus it is a cell surface protein. Its extracellular domain is at residues 1-685. Endosialin may be involved in endocytosis. The mouse ortholog is predicted to have a signal peptide at residues 1-21.
- [57] TEM 2 is a dexamethasone induced, ras related protein homolog of 266 amino acids. It has neither a signal sequence nor a transmembrane domain. Thus it is neither a cell surface nor a secreted protein. TEM 2 plays a role in signal transduction. It regulates alterations in cell morphology, proliferation, and cell-extracellular matrix interactions.
- [58] TEM 3 (originally termed TEM 7R) has both a signal sequence (at residues 1-24 or 1-30) and a transmembrane domain (at residues 456 - 477). Thus it is a cell surface protein. The portion of the protein which is extracellular is at amino acids 1- 455. TEM 3 has domains with homology to integrins, plexin, and adhesion molecules. TEM 3 may regulate GTPases that control signal transduction pathways linking plasma membrane receptors to

the actin cytoskeleton. In the mouse ortholog, the signal peptide is predicted to be residues 1-30.

[59] TEM 4 is also known as DKK-3. It has a signal sequence (residues 1-16), suggesting that it is a secreted protein. TEM 4 regulates *wnt* signaling, and it may be involved in vasculogenesis and *wnt*-dependent signaling for endothelial growth. TEM 4 is an inhibitor of Wnt oncogene and such inhibition can be determined by assay. Tsuji et al., *Biochem.Biophys.Res.Comm.* 268:20-4, 2000.

[60] TEM 5 appears to be neither secreted nor a cell surface protein. TEM 5 appears to be a component of a G protein - GTPase signaling pathway.

[61] TEM 6 is also known as stromelysin - 3 /Matrix metalloproteinase 11 (MMP -11). It has a signal sequence at residues 1-31, but no transmembrane domain. It has an alternative signal peptide splice site at residues 108-109. Thus it appears to be a secreted protein. TEM 6 belongs to the zinc metalloprotease family, also known as the matrixin subfamily. TEM 6 is expressed in most invasive carcinomas. Alpha 1 - protease inhibitor is a natural substrate of MMP 11. TEM 6 degrades extracellular matrix proteins such as collagen and is involved in extracellular matrix remodeling and cell migration. Stromelysin can be assayed using a casein-resorufin substrate, for example. See Tortorella and Amer, *Inflammation Research* 46 Supp. 2:S122-3, 1997.

[62] TEM 7 is a protein of many names, also being known as matrix metalloprotease 2, gelatinase A, and 72KD type IV collagenase. TEM 7 has a signal sequence at residues 1-26 and is a secreted protein. Like TEM 6, TEM 7 belongs to the matrixin subfamily (zinc metalloproteinases). TEM 7 cleaves gelatin type I, collagen type I, IV, V VII and X. TEM 7 associates with integrin on the surface of endothelial cells and promotes vascular invasion. TEM 7 is involved in tissue remodeling. TEM 7 can be assayed using zymography or quenched fluorescent substrate hydrolysis, for example.

Garbett, et al., Molecular Pathology 53:99-106, 2000. A fluorogenic matrix metalloproteinase substrate assay can also be used which employs methoxycoumarin containing septapeptide analog of the alpha2(I) collagen cleavage site. See Bhide et al., J. Periodontology 71:690-700, 2000.

- [63] TEM 8 is HEYL protein. It has neither a signal sequence nor a transmembrane domain. It is related to the hairy/Enhancer of split genes. TEM 8 is likely a nuclear protein, having a role as a transcription factor. TEM 8 belongs to a new class of Notch signal transducers and plays a key role in various developmental processes, such as vascular development, somatogenesis and neurogenesis. SNP's at residues 615 and 2201 have Cytosine bases. Notch 3 mutations underlie the CADASIL vascular disorder. See *Mech Dev* 2000 Nov; 98 (1-2):175

- [64] TEM 9 is a G- protein coupled receptor homolog, having both a signal sequence at residues 1-26 and 7 transmembrane domains. Thus it is a cell surface protein. Its extracellular region resides in amino acids 1-769. Its transmembrane domains are at residues 817-829 (TM2 and TM3), residues 899-929 (TM4 and TM5), and residues 1034-1040 (TM6 and TM7). TEM 9 acts as a G-protein coupled receptor with extracellular domains characteristic of cell adhesion proteins. One of its splice variants may function as a soluble receptor. TEM 9 may regulate cell polarity and cell migration. It may be involved in exocytosis based on latrophilin function. The mouse ortholog has a predicted signal peptide at residues 1-29.

- [65] TEM 10 is collagen type I, alpha2 (COL1A2), which has a signal sequence at residues 1-22. It is an extracellular matrix (ECM) protein which is secreted subsequent to synthesis. TEM 10 interacts with a number of proteins including other ECM proteins, certain growth factors, and matrix metalloproteases. TEM 10 is required for the induction of endothelial tube formation and is involved in tissue remodeling. A variant at nucleotide 3233 which substitutes an A, is associated with osteogenesis imperfecta type IV. A variant at nucleotide 4321 substituting an A retains a wild type phenotype.

Nucleotide 715 is a site of a polymorphism. Nucleotides 695-748 are deleted in Ehlers-Danos syndrome. Other mutations are associated with idiopathic osteoporosis, and atypical Marfan syndrome. Variants are known at nucleotides 226(T,C), 314(A,C), 385(T,C), 868 (G,A), 907(C,T), 965(A,G), 970(T,A), 1784 (G,C), 2017(T,G), 2172(C,A), 2284(T,C), 2308(T,C), 2323(T,G), 2344(T,G), 2604(G,A), 2974(A,T), 2903(A,G), 2995(C,T), 3274(C,T), 3581(A,C), 3991(A,C), 4201(G,T), 4434(C,T), 4551(A,C), 4606(C,A), 4947(T,C), 4978(C,T), 4982(G,T), 5051(G,T). PolyA sites are located at nucleotides 4450, 4550, 4885, and 5082. PolyA signals are located at 4420-4424, 4515-4520, 4529-4534, 4866-4871, 5032-5037, 5053-5058. TEM 10, 20, and 40 derive from the same gene but are different isoforms having different lengths.

- [66] TEM 11 is Nidogen /Entactin. It is a secreted protein which has a signal sequence at residues 1-28. TEM 11 is an extracellular matrix protein which is a component of a basement membrane. TEM 11 binds to laminin and collagen IV and other extracellular matrix proteins. TEM 11 regulates capillary formation and is involved in tissue remodelling. Variations have been observed at nucleotides 4265(T,C), 4267(G,C,T), and 4738(T,G). Nidogen can be assayed by its effect on the morphology of astrocytes. See Grimpe et al., GLIA 28:138-49, 1999.

- [67] TEM 12 is the alpha 3 chain of collagen type VI. It has a signal sequence at residues 1-25. A secreted protein, TEM 12 is an extracellular matrix protein. TEM 12 has a splice variant. TEM 12 is a major constituent of vascular subendothelium and is involved in tissue remodeling. It regulates platelet activation and aggregation. Alternatively spliced domains are located at nucleotides 347-964, 965-1567, 2153-3752, and 4541-5041.

- [68] TEM 13 is also known as Thy -1 glycoprotein. It has both a signal sequence (at residues 1-19) and a transmembrane domain (at residues 143-159). Residues 131-161 are removed in a matured form of the protein. The extracellular region of the protein is residues 1-142 or residues 1-130. TEM

13 has a glycosyl phosphatidylinositol (GPI) anchor at residue 130 anchoring it to the membrane. TEM 13 is detectable in its soluble form in human serum. TEM 13 is reported to be a marker for activated endothelial cells (a marker of adult but not embryonic angiogenesis). TEM 13 on vascular endothelial cells may function as a possible vascular permeability modulator. Antibody to Thy-1 is a mitogenic signal for the CD4+CD45+ and CD8+CD45+ cells, but fails to induce proliferation in the CD45- T cells. Pingel et al., International Immunology 6:169-78, 1994. Thy-1 can be assayed as an inhibitor of such signal.

[69] TEM 14 is also known as cystatin S. It is a secreted protein with a signal sequence at residues 1-20 and an extracellular region at residues 1-141. It is a cysteine protease inhibitor. TEM 14 may regulate cysteine protease function involved in angiogenesis and tissue remodeling. TEM14 is an inhibitor of the activity of papain and such inhibition can be assayed. Hiltke et al., J. Dental Research 78:1401-9, 1999.

[70] TEM 15 is collagen type III, alpha 1 (COL3A1). It has a signal sequence (residues 1-23) and is secreted. Type III collagen binds to von Willebrand factor. It is involved in cell-cell adhesion, proliferation, and migration activities. Variants at nucleotides 2104(C,A), 2194(G,A), 2346(C,T), 2740(C,T), 3157(T), 3468(G), 3652(T), 3666(C), 3693(C), 3755(G), 3756(T), 3824(C), 4546(A, G), 4661(G), 4591(C,T), 4665(C), 5292(C), 5293(C), and 5451 (A) have been observed.

[71] TEM 16 is a tensin homolog which is apparently an intracellular protein. It may have splice variants or isoforms. One form with 1704 amino acids has a region at the N-terminal domain which is similar to a tumor suppressor protein, phosphatase and tensin homolog (PTEN). Tensin is a focal adhesion molecule that binds to actins and phosphorylated proteins. It is involved in cell migration linking signal transduction pathways to the cytoskeleton. PTEN regulates tumor induced angiogenesis.

[72] TEM 17 (BSC-TEM 7) has a signal sequence which includes residues 1-18 and a transmembrane domain at residues 427-445. It is a cell surface marker with an extracellular region comprising residues 1-426. It has homologs in both mouse and *C. elegans*. Residues 137-244 share weak homology with nidogen; residues 280-344 share homology to PSI domains found in plexin, semaphorins and integrin beta subunits. Variants have been observed at nucleotides 1893(A,G), 1950(C,G), 2042(A,G), and 2220(G,A). In mouse TEM 17 the signal sequence includes residues 1-19.

[73] TEM 19 was originally reported to be tumor endothelial marker 8, *i.e.*, BSC-TEM 8. It has a signal sequence at residues 1-27 and a transmembrane domain at residues 322-343. It is a cell surface protein having an extracellular region at residues 1-321. TEM 19 has a von Willebrand Factor (vWF) A domain at residues 44-216; a domain at residues 34-253 which is found in leukointegrin alpha D chain; and a domain at residues 408-560 found in PRAM-1 or adaptor molecule -1 of the vinculin family. TEM 19's function is adhesion related. von Willebrand Factor domains are typically involved in a variety of functions including vascular processes. TEM 19 may play a role in the migration of vascular endothelial cells. The mouse ortholog has a predicted signal peptide at residues 1-27.

[74] TEM 20 is collagen type I, alpha 2 (COL1A2). It has a signal sequence at residues 1-22 and is a secreted extracellular matrix protein. TEM 20 induces endothelial tube formation *in vitro* and is involved in tissue remodeling. Variants have been observed at nucleotides 226(T,C), 314(A,C), 385(T,C), 868 (G,A), 907(C,T), 965(A,G), 970(T,A), 1784(G,C), 2017(T,G), 2172(C,A), 2284(T,C), 2308(T,C), 2323(T,G), 2344(T,G), 2604(G,A), 2794(A,T), 2903(A,G), 2995(C,T), 3274(C,T), 3581(A,C), 3991(A,C), 4201(G,T), 4434(C,T), 4551(A,C), 4606(C,A), 4895-4901(-, GGACAAC), 4947(T,C), 4978(C,T), 4982(G,T), 5051(G,T).

[75] TEM 21 is a Formin - like protein homolog which is an intracellular protein. Formin related proteins interact with Rho family small GTPases,

profilin, and other actin associated proteins. Formin-binding proteins bind to FHL domains with their WW domains. TEM 21 has a proline rich FHL domain at residues 221-449. Formin related proteins play crucial roles in morphogenesis, cell polarity, cytokinesis and reorganization of the actin cytoskeleton. They may also regulate apoptosis, cell adhesion and migration.

- [76] TEM 22 is an endocytic receptor in the macrophage mannose receptor family. It has both a signal sequence at residues 1-30 and a transmembrane domain at residues 1415-1435, and resides on the cell surface. Its extracellular domain is amino acids 1- 1414. TEM 22 may be present as a soluble (secreted) form and act as an inhibitor. It may bind secreted phospholipase A2 (sPLA2) and mediate biological responses elicited by sPLA2. TEM 22 may have endocytic properties for sPLA2 and mediate endocytosis for endothelial related proteins. It may promote cell adhesion and be involved in cell-cell communication. Variations have been observed at nucleotide 5389 (A, G). TEM 22 mediates uptake of micro-organisms and host-derived glycoproteins. Groger et al., J. Immunology 165:5428-34, 2000.

- [77] TEM 24 is tensin, an intracellular protein. It is a focal adhesion molecule that binds to actin filaments and interacts with phosphotyrosine containing proteins. It may mediate kinase signaling activities and regulate cellular transformation. Variations have been observed at nucleotides 2502 (A, G), 2622(A, G), 6027(A, G). TEM24 binds to actin filaments and interacts with phosphotyrosine-containing proteins. Chen et al., Biochem. J. 351 Pt2:403-11, 2000. TEM24 also binds to phosphoinositide3-kinase. Auger et al., J. Bio. Chem. 271:23452-7, 1996 TEM 24 also binds to nuclear protein p130. Lo et al., Bioessays 16:817-23, 1994.

- [78] TEM 25 is Bone morphogenic protein 1 (BMP-1) which has a signal sequence at residues 1-22. It is a secreted protein. There are at least 6 isoforms of BMP-1 as well as splice variants which add carboxy terminal CUB domains and an additional EGF domain. TEM 25 is a metalloprotease enzyme. It cleaves the C-terminal propeptide of collagen type I, II and III and

laminin 5 gamma 2, proteins that are important for vascular processes. It is involved in cartilage formation. Variations have been observed at nucleotides 3106(C,T), 3248(G,A), 3369(G,A). TEM 25 cleave probiglycan at a single site, removing the propeptide and producing a biglycan molecule with an NH(2) terminus identical to that of the mature form found in tissues. Scft et al., J. Biol. Chem. 275:30504-11, 2000. Laminin alpha 3 and gamma2 short chains are substrates of TEM 25. Amano et al., J. Biol. Chem. 275:22728-35, 2000.

[79] TEM 27 is known as Slit homolog 3, a secreted protein with a signal sequence at residues 1-27. TEM 27 is a secreted guide protein involved in migration, repulsion and patterning. It interacts with "round about" receptors (Robo receptors). TEM 27 may interact with extracellular matrix (ECM) proteins and is involved in cell adhesion. Variations have been observed at nucleotides 4772 (C,T)

[80] TEM 28 is similar to mouse nadrin (neuron specific GTPase activating protein). TEM 28 is an intracellular protein with a RhoGAP domain. The RhoGAP domain activates RhoA, Rac1, and Cdc42 GTPases. It is involved in the reorganization of actin filaments and enhancing exocytosis. It may also be involved in cell signalling. Variations have been observed at nucleotide 3969 (A,C),

[81] TEM 29 is protein tyrosine phosphatase type IVA, member 3, isoform 1, an intracellular protein. It has alternate splice variants. TEM 29 belongs to a small class of prenylated protein tyrosine phosphatases (PTPs). It may be membrane associated by prenylation. PTPs are cell signaling molecules and play regulatory roles in a variety of cellular processes and promote cell proliferation. PTP PRL-3 regulates angiotensin-II induced signaling events.

[82] TEM 30 is integrin alpha 1, a cell surface protein having both a signal sequence (residues 1-28) and a transmembrane domain (residues 1142- 1164). Its extracellular region includes amino acids 1-1141. TEM 30 is a receptor for

laminin and collagen. It mediates a variety of adhesive interactions. TEM 30 is abundantly expressed on microvascular endothelial cells. It stimulates endothelial cell proliferation and vascularization. TEM 30 may regulate angiostatin production. Variations have been observed at nucleotide 418 (C,T). TEM 30 activates the Ras/Shc/mitogen-activated protein kinase pathway promoting fibroblast cell proliferation. It also acts to inhibit collagen and metalloproteinase synthesis. Pozzi et al., Proc. Nat. Acad. Sci. USA 97:2202-7, 2000,

- [83] TEM 31 is Collagen IV alpha 1 (COL4A1) a secreted protein with a at residues 1-27. TEM 31 is a component of the basement membrane. It binds to alpha3 beta 1 integrin and promotes integrin mediated cell adhesion. Non-collagenous domains of type IV subunits are involved in tumoral angiogenesis. TEM 31 is involved in tissue remodeling. Variations have been observed at nucleotide 4470 (C,T)

- [84] TEM 33 is methylmalonyl Co-A Mutase a protein which is localized in the mitochondrial matrix. It degrades several amino acids, odd-numbered-acid fatty acids, and cholesterol to the tricarboxylic acid cycle. A defect in TEM 33 causes a fatal disorder in organic acid metabolism termed methylmalonic aciduria. Variations have been observed at nucleotides 1531(G,A), 1671(G,A), 2028(T,C), 2087(G,A), 2359(A,G), 2437(C,A), 2643(G,C), 2702(G,C). TEM 33 converts L-methylmalonyl CoA to succinyl CoA. This reaction can be assayed as is known in the art. See, e.g., Clin. Chem. 41(8 Pt D):1164-70, 1995.

- [85] TEM 36 is collagen type XII, alpha1 (COL12A1) , an extracellular matrix protein having a signal sequence at residues 1-23 or 24. TEM 36 has von Willebrand Factor (vWF) type A domains, Fibronectin type III domains, and thrombospondin N-terminal like domain. TEM 36 is expressed in response to stress environment. TEM 36 may organize extracellular matrix architecture and be involved in matrix remodeling. There are two isoforms of the protein, a long form and a short form. The short form is missing amino acids 25-1188,

and therefore nucleotides 73 to 3564. Both forms share the signal sequence and are therefore both secreted.

- [86] TEM 37 is lumican, an extracellular matrix sulfated proteoglycan having a signal sequence at residues 1-18. Lumican interacts with proteins that are involved in matrix assembly such as collagen type I and type VI; it is involved in cell proliferation and tissue morphogenesis. Lumican plays an important role in the regulation of collagen fiber assembly. Variations have been observed at nucleotides 1021(G,T), 1035(A,G), 1209(A,G), 1259(A,C), 1418(C,A), 1519(T,A). TEM 37 is a binding partner of TGF- β . See FASEB J. 15:559-61, 2000. One assay that can be used to determine TEM 37 activity is a collagen fibril formation/sedimentation assay. Svensson et al, FEBS Letters 470:178-82, 2000.
- [87] TEM 38 is collagen type I, alpha 1 (COL1A1), an extracellular matrix protein having a signal sequence at residues 1-22. Type I collagen promotes endothelial cell migration and vascularization and induces tube formation and is involved in tissue remodelling. Telopeptide derivative is used as a marker for malignancy and invasion for certain cancer types. Variations have been observed at nucleotides 296(T,G), 1810(G,A), 1890(G,A), 2204(T,A), 3175(G,C), 3578(C,T), 4298(C,T), 4394(A,T), 4410(A,C), 4415(C,A), 4419(A,T), 4528(C,A), 4572(G,T), 4602(T,C), 5529(T,C), 5670(C,T), 5985(C,T), 6012(C,T).
- [88] TEM 39 is transforming growth factor β -3 (TGF-beta3). It has a signal sequence at residues 1-23. It is a secreted protein. TEM 39 regulates cell growth and differentiation. TGF-beta isoforms play a major role in vascular repair processes and remodeling. Variations have been observed at nucleotide 2020(G,T).
- [89] TEM 41 is similar to Olfactomedin like protein. It appears to be an intracellular protein, having no obvious predicted signal sequence. Olfactomedin is the major glycoprotein of the extracellular mucous matrix of

olfactory neuroepithelium. TEM 41 shares homology with latrophilin (extracellular regions) which has cell-adhesive type domains. TEM 41 may be involved in adhesive function.

[90] TEM 42 is MSTP032 protein, a cell surface protein having a transmembrane domain at residues 42-61. Its function is unknown and it shares little homology with other proteins. Variations have been observed at nucleotides 418(A,T), 724(C,A).

[91] TEM 44 is a hypothetical protein FLJ11190 (NM_018354) which has two predicted transmembrane domains at residues 121-143 and 176-197. Residues 144-175 may form an extracellular region. TEM 44's function is not known and shares no homology to other known proteins.

[92] TEM 45 is tropomyosin 1 (alpha), a protein which is intracellular. It forms dimers with a beta subunit. It influences actin function. TEM 45 may be involved in endothelial cell cytoskeletal rearrangement. Variations have been observed at nucleotides 509(A,C), 621(A,C), 635(T,G), 642(C,G), 1059(G,T).

[93] TEM 46 is peanut-like 1 protein/septin 5, which belongs to the septin family. Proteins in the septin family bind to GTP and phosphatidylinositol 4,5-bisphosphate. They are involved in the signal transduction cascades controlling cytokinesis and cell division.

[94] NEM 4 is a member of the small inducible cytokine subfamily A (cys-cys), member 14 (SCYA14). NEM4 is a secreted protein characterized by two adjacent cysteine residues. One isoform lacks internal 16 amino acids compared to isoform 2.

[95] NEM 22 shares homology with guanylate kinase-interacting protein 1(Maguin-1. It is a membrane associated protein.

- [96] NEM 23 is human signaling lymphocytic activation molecule (SLAM). It has a signal sequence at residues 1-20. The extracellular domain may reside at residues 21-237. There is a secreted isoform of the protein.
- [97] NEM33 is netrin 4. It induces neurite outgrowth and promotes vascular development. At higher concentration, neurite outgrowth is inhibited.
- [98] ECs represent only a minor fraction of the total cells within normal or tumor tissues, and only those EC transcripts expressed at the highest levels would be expected to be represented in libraries constructed from unfractionated tissues. The genes described in the current study should therefore provide a valuable resource for basic and clinical studies of human angiogenesis in the future. Genes which have been identified as tumor endothelial markers (TEMs) correspond to tags shown in SEQ ID NOS: 94-139, 173-176, 180-186. Genes which have been identified as normal endothelial markers (NEMs) correspond to tags shown in SEQ ID NOS: 140-172. Genes which have been identified as pan-endothelial markers (PEMs) *i.e.*, expressed in both tumor and normal endothelial cells correspond to tags shown in SEQ ID NOS: 1-93. Genes which have been previously identified as being expressed predominantly in the endothelium correspond to PEM tags shown in SEQ ID NOS: 1-6, 8, 10-15. Markers in each class can be used interchangeably for some purposes.
- [99] Isolated and purified nucleic acids, according to the present invention are those which are not linked to those genes to which they are linked in the human genome. Moreover, they are not present in a mixture such as a library containing a multitude of distinct sequences from distinct genes. They may be, however, linked to other genes such as vector sequences or sequences of other genes to which they are not naturally adjacent. Tags disclosed herein, because of the way that they were made, represent sequences which are 3' of the 3' most restriction enzyme recognition site for the tagging enzyme used to generate the SAGE tags. In this case, the tags are 3' of the most 3' most NlaIII site in the cDNA molecules corresponding to mRNA. Nucleic acids corresponding to tags may be RNA, cDNA, or genomic DNA, for example.

Such corresponding nucleic acids can be determined by comparison to sequence databases to determine sequence identities. Sequence comparisons can be done using any available technique, such as BLAST, available from the National Library of Medicine, National Center for Biotechnology Information. Tags can also be used as hybridization probes to libraries of genomic or cDNA to identify the genes from which they derive. Thus, using sequence comparisons or cloning, or combinations of these methods, one skilled in the art can obtain full-length nucleic acid sequences. Genes corresponding to tags will contain the sequence of the tag at the 3' end of the coding sequence or of the 3' untranslated region (UTR), 3' of the 3' most recognition site in the cDNA for the restriction endonuclease which was used to make the tags. The nucleic acids may represent either the sense or the anti-sense strand. Nucleic acids and proteins although disclosed herein with sequence particularity, may be derived from a single individual. Allelic variants which occur in the population of humans are including within the scope of such nucleic acids and proteins. Those of skill in the art are well able to identify allelic variants as being the same gene or protein. Given a nucleic acid, one of ordinary skill in the art can readily determine an open reading frame present, and consequently the sequence of a polypeptide encoded by the open reading frame and, using techniques well known in the art, express such protein in a suitable host. Proteins comprising such polypeptides can be the naturally occurring proteins, fusion proteins comprising exogenous sequences from other genes from humans or other species, epitope tagged polypeptides, etc. Isolated and purified proteins are not in a cell, and are separated from the normal cellular constituents, such as nucleic acids, lipids, etc. Typically the protein is purified to such an extent that it comprises the predominant species of protein in the composition, such as greater than 50, 60, 70, 80, 90, or even 95% of the proteins present.

[100] Using the proteins according to the invention, one of ordinary skill in the art can readily generate antibodies which specifically bind to the proteins. Such antibodies can be monoclonal or polyclonal. They can be chimeric, humanized, or totally human. Any functional fragment or derivative of an

antibody can be used including Fab, Fab', Fab2, Fab'2, and single chain variable regions. So long as the fragment or derivative retains specificity of binding for the endothelial marker protein it can be used. Antibodies can be tested for specificity of binding by comparing binding to appropriate antigen to binding to irrelevant antigen or antigen mixture under a given set of conditions. If the antibody binds to the appropriate antigen at least 2, 5, 7, and preferably 10 times more than to irrelevant antigen or antigen mixture then it is considered to be specific.

- [101] Techniques for making such partially to fully human antibodies are known in the art and any such techniques can be used. According to one particularly preferred embodiment, fully human antibody sequences are made in a transgenic mouse which has been engineered to express human heavy and light chain antibody genes. Multiple strains of such transgenic mice have been made which can produce different classes of antibodies. B cells from transgenic mice which are producing a desirable antibody can be fused to make hybridoma cell lines for continuous production of the desired antibody. See for example, Nina D. Russel, Jose R. F. Corvalan, Michael L. Gallo, C. Geoffrey Davis, Liise-Anne Pirofski. Production of Protective Human Antipneumococcal Antibodies by Transgenic Mice with Human Immunoglobulin Loci *Infection and Immunity* April 2000, p. 1820-1826; Michael L. Gallo, Vladimir E. Ivanov, Aya Jakobovits, and C. Geoffrey Davis. The human immunoglobulin loci introduced into mice: V (D) and J gene segment usage similar to that of adult humans *European Journal of Immunology* 30: 534-540, 2000; Larry L. Green. Antibody engineering via genetic engineering of the mouse: XenoMouse strains are a vehicle for the facile generation of therapeutic human monoclonal antibodies *Journal of Immunological Methods* 231 11-23, 1999; Yang X-D, Corvalan JRF, Wang P, Roy CM-N and Davis CG. Fully Human Anti-interleukin-8 Monoclonal Antibodies: Potential Therapeutics for the Treatment of Inflammatory Disease States. *Journal of Leukocyte Biology* Vol. 66, pp401-410 (1999); Yang X-D, Jia X-C, Corvalan JRF, Wang P, CG Davis and Jakobovits A. Eradication of Established Tumors by a Fully Human Monoclonal Antibody to the Epidermal

Growth Factor Receptor without Concomitant Chemotherapy. *Cancer Research* Vol. 59, Number 6, pp1236-1243 (1999) ; Jakobovits A. Production and selection of antigen-specific fully human monoclonal antibodies from mice engineered with human Ig loci. *Advanced Drug Delivery Reviews* Vol. 31, pp: 33-42 (1998); Green L and Jakobovits A. Regulation of B cell development by variable gene complexity in mice reconstituted with human immunoglobulin yeast artificial chromosomes. *J. Exp. Med.* Vol. 188, Number 3, pp: 483-495 (1998); Jakobovits A. The long-awaited magic bullets: therapeutic human monoclonal antibodies from transgenic mice. *Exp. Opin. Invest. Drugs* Vol. 7(4), pp : 607-614 (1998) ; Tsuda H, Maynard-Currie K, Reid L, Yoshida T, Edamura K, Maeda N, Smithies O, Jakobovits A. Inactivation of Mouse HPRT locus by a 203-bp retrotransposon insertion and a 55-kb gene-targeted deletion: establishment of new HPRT-Deficient mouse embryonic stem cell lines. *Genomics* Vol. 42, pp: 413-421 (1997) ; Sherman-Gold, R. Monoclonal Antibodies: The Evolution from '80s Magic Bullets To Mature, Mainstream Applications as Clinical Therapeutics. *Genetic Engineering News* Vol. 17, Number 14 (August 1997); Mendez M, Green L, Corvalan J, Jia X-C, Maynard-Currie C, Yang X-d, Gallo M, Louie D, Lee D, Erickson K, Luna J, Roy C, Abderrahim H, Kirschenbaum F, Noguchi M, Smith D, Fukushima A, Hales J, Finer M, Davis C, Zsebo K, Jakobovits A. Functional transplant of megabase human immunoglobulin loci recapitulates human antibody response in mice. *Nature Genetics* Vol. 15, pp: 146-156 (1997); Jakobovits A. Mice engineered with human immunoglobulin YACs: A new technology for production of fully human antibodies for autoimmunity therapy. *Weir's Handbook of Experimental Immunology, The Integrated Immune System* Vol. IV, pp: 194.1-194.7 (1996) ; Jakobovits A. Production of fully human antibodies by transgenic mice. *Current Opinion in Biotechnology* Vol. 6, No. 5, pp: 561-566 (1995) ; Mendez M, Abderrahim H, Noguchi M, David N, Hardy M, Green L, Tsuda H, Yeast S, Maynard-Currie C, Garza D, Gemmill R, Jakobovits A, Klapholz S. Analysis of the structural integrity of YACs comprising human immunoglobulin genes in yeast and in embryonic stem cells. *Genomics* Vol. 26, pp: 294-307 (1995); Jakobovits A. YAC Vectors: Humanizing the mouse genome. *Current Biology* Vol. 4, No. 8, pp:

761-763 (1994); Arbones M, Ord D, Ley K, Ratech H, Maynard-Curry K, Otten G, Capon D, Tedder T. Lymphocyte homing and leukocyte rolling and migration are impaired in L-selectin-deficient mice. *Immunity* Vol. 1, No. 4, pp: 247-260 (1994); Green L, Hardy M, Maynard-Curry K, Tsuda H, Louie D, Mendez M, Abderrahim H, Noguchi M, Smith D, Zeng Y, et. al. Antigen-specific human monoclonal antibodies from mice engineered with human Ig heavy and light chain YACs. *Nature Genetics* Vol. 7, No. 1, pp: 13-21 (1994); Jakobovits A, Moore A, Green L, Vergara G, Maynard-Curry K, Austin H, Klapholz S. Germ-line transmission and expression of a human-derived yeast artificial chromosome. *Nature* Vol. 362, No. 6417, pp: 255-258 (1993) ; Jakobovits A, Vergara G, Kennedy J, Hales J, McGuinness R, Casentini-Borocz D, Brenner D, Otten G. Analysis of homozygous mutant chimeric mice: deletion of the immunoglobulin heavy-chain joining region blocks B-cell development and antibody production. *Proceedings of the National Academy of Sciences USA* Vol. 90, No. 6, pp: 2551-2555 (1993); Kucheralapati et al., U.S. 6,1075,181.

[102] Antibodies can also be made using phage display techniques. Such techniques can be used to isolate an initial antibody or to generate variants with altered specificity or avidity characteristics. Single chain Fv can also be used as is convenient. They can be made from vaccinated transgenic mice, if desired. Antibodies can be produced in cell culture, in phage, or in various animals, including but not limited to cows, rabbits, goats, mice, rats, hamsters, guinea pigs, sheep, dogs, cats, monkeys, chimpanzees, apes.

[103] Antibodies can be labeled with a detectable moiety such as a radioactive atom, a chromophore, a fluorophore, or the like. Such labeled antibodies can be used for diagnostic techniques, either *in vivo*, or in an isolated test sample. Antibodies can also be conjugated, for example, to a pharmaceutical agent, such as chemotherapeutic drug or a toxin. They can be linked to a cytokine, to a ligand, to another antibody. Suitable agents for coupling to antibodies to achieve an anti-tumor effect include cytokines, such as interleukin 2 (IL-2) and Tumor Necrosis Factor (TNF); photosensitizers, for

use in photodynamic therapy, including aluminum (III) phthalocyanine tetrasulfonate, hematoporphyrin, and phthalocyanine; radionuclides, such as iodine-131 (^{131}I), yttrium-90 (^{90}Y), bismuth-212 (^{212}Bi), bismuth-213 (^{213}Bi), technetium-99m ($^{99\text{m}}\text{Tc}$), rhenium-186 (^{186}Re), and rhenium-188 (^{188}Re); antibiotics, such as doxorubicin, adriamycin, daunorubicin, methotrexate, daunomycin, neocarzinostatin, and carboplatin; bacterial, plant, and other toxins, such as diphtheria toxin, pseudomonas exotoxin A, staphylococcal enterotoxin A, abrin-A toxin, ricin A (deglycosylated ricin A and native ricin A), TGF-alpha toxin, cytotoxin from chinese cobra (*Naja naja atra*), and gelonin (a plant toxin); ribosome inactivating proteins from plants, bacteria and fungi, such as restrictocin (a ribosome inactivating protein produced by *Aspergillus restrictus*), saporin (a ribosome inactivating protein from *Saponaria officinalis*), and RNase; tyrosine kinase inhibitors; ly207702 (a difluorinated purine nucleoside); liposomes containing antitumor agents (*e.g.*, antisense oligonucleotides, plasmids which encode for toxins, methotrexate, etc.); and other antibodies or antibody fragments, such as F(ab).

[104] Those of skill in the art will readily understand and be able to make such antibody derivatives, as they are well known in the art. The antibodies may be cytotoxic on their own, or they may be used to deliver cytotoxic agents to particular locations in the body. The antibodies can be administered to individuals in need thereof as a form of passive immunization.

[105] Characterization of extracellular regions for the cell surface and secreted proteins from the protein sequence is based on the prediction of signal sequence, transmembrane domains and functional domains. Antibodies are preferably specifically immunoreactive with membrane associated proteins, particularly to extracellular domains of such proteins or to secreted proteins. Such targets are readily accessible to antibodies, which typically do not have access to the interior of cells or nuclei. However, in some applications, antibodies directed to intracellular proteins may be useful as well. Moreover, for diagnostic purposes, an intracellular protein may be an equally good target since cell lysates may be used rather than a whole cell assay.

- [106] Computer programs can be used to identify extracellular domains of proteins whose sequences are known. Such programs include SMART software (Schultz et al., Proc. Natl. Acad. Sci. USA 95: 5857-5864, 1998) and Pfam software (Bateman et al., Nucleic acids Res. 28: 263-266, 2000) as well as PSORTII. Typically such programs identify transmembrane domains; the extracellular domains are identified as immediately adjacent to the transmembrane domains. Prediction of extracellular regions and the signal cleavage sites are only approximate. It may have a margin of error + or - 5 residues. Signal sequence can be predicted using three different methods (Nielsen et al, *Protein Engineering* 10: 1-6 ,1997, Jagla et. al, *Bioinformatics* 16: 245-250 , 2000, Nakai, K and Horton, P. Trends in Biochem. Sci. 24:34-35, 1999) for greater accuracy. Similarly transmembrane (TM) domains can be identified by multiple prediction methods. (Pasquier, et. al, *Protein Eng.* 12:381-385, 1999, Sonnhammer et al., In Proc. of Sixth Int. Conf. on Intelligent Systems for Molecular Biology, p. 175-182 , Ed J. Glasgow, T. Littlejohn, F. Major, R. Lathrop, D. Sankoff, and C. Sensen Menlo Park, CA: AAAI Press, 1998 , Klein, et.al, *Biochim. Biophys. Acta*, 815:468, 1985, Nakai and Kanehisa *Genomics*, 14: 897-911 , 1992). In ambiguous cases, locations of functional domains in well characterized proteins are used as a guide to assign a cellular localization.
- [107] Putative functions or functional domains of novel proteins can be inferred from homologous regions in the database identified by BLAST searches (Altschul et. al. *Nucleic Acid Res.* 25: 3389-3402, 1997) and/or from a conserved domain database such as Pfam (Bateman et.al, *Nucleic Acids Res.* 27:260-262 1999) BLOCKS (Henikoff, et. al, *Nucl. Acids Res.* 28:228-230, 2000) and SMART (Ponting, et. al, *Nucleic Acid Res.* 27:229-232, 1999). Extracellular domains include regions adjacent to a transmembrane domain in a single transmembrane domain protein (out-in or type I class). For multiple transmembrane domains proteins, the extracellular domain also includes those regions between two adjacent transmembrane domains (in-out and out-in). For type II transmembrane domain proteins, for which the N-terminal region is cytoplasmic, regions following the transmembrane domain is generally

extracellular. Secreted proteins on the other hand do not have a transmembrane domain and hence the whole protein is considered as extracellular.

[108] Membrane associated proteins can be engineered to delete the transmembrane domains, thus leaving the extracellular portions which can bind to ligands. Such soluble forms of transmembrane receptor proteins can be used to compete with natural forms for binding to ligand. Thus such soluble forms act as inhibitors, and can be used therapeutically as anti-angiogenic agents, as diagnostic tools for the quantification of natural ligands, and in assays for the identification of small molecules which modulate or mimic the activity of a TEM:ligand complex.

[109] Alternatively, the endothelial markers themselves can be used as vaccines to raise an immune response in the vaccinated animal or human. For such uses, a protein, or immunogenic fragment of such protein, corresponding to the intracellular, extracellular or secreted TEM of interest is administered to a subject. The immunogenic agent may be provided as a purified preparation or in an appropriately expressing cell. The administration may be direct, by the delivery of the immunogenic agent to the subject, or indirect, through the delivery of a nucleic acid encoding the immunogenic agent under conditions resulting in the expression of the immunogenic agent of interest in the subject. The TEM of interest may be delivered in an expressing cell, such as a purified population of tumor endothelial cells or a populations of fused tumor endothelial and dendritic cells. Nucleic acids encoding the TEM of interest may be delivered in a viral or non-viral delivery vector or vehicle. Non-human sequences encoding the human TEM of interest or other mammalian homolog can be used to induce the desired immunologic response in a human subject. For several of the TEMs of the present invention, mouse, rat or other ortholog sequences are described herein or can be obtained from the literature or using techniques well within the skill of the art.

[110] Endothelial cells can be identified using the markers which are disclosed herein as being endothelial cell specific. These include the human markers

identified by SEQ ID NOS: 1-172, *i.e.*, the normal, pan-endothelial, and the tumor endothelial markers. Homologous mouse markers include tumor endothelial markers of SEQ ID NO: 182-186 and 190-194. Antibodies specific for such markers can be used to identify such cells, by contacting the antibodies with a population of cells containing some endothelial cells. The presence of cross-reactive material with the antibodies identifies particular cells as endothelial. Similarly, lysates of cells can be tested for the presence of cross-reactive material. Any known format or technique for detecting cross-reactive material can be used including, immunoblots, radioimmunoassay, ELISA, immunoprecipitation, and immunohistochemistry. In addition, nucleic acid probes for these markers can also be used to identify endothelial cells. Any hybridization technique known in the art including Northern blotting, RT-PCR, microarray hybridization, and in situ hybridization can be used.

- [111] One can identify tumor endothelial cells for diagnostic purposes, testing cells suspected of containing one or more TEMs. One can test both tissues and bodily fluids of a subject. For example, one can test a patient's blood for evidence of intracellular and membrane associated TEMs, as well as for secreted TEMs. Intracellular and/or membrane associated TEMs may be present in bodily fluids as the result of high levels of expression of these factors and/or through lysis of cells expressing the TEMs.
- [112] Populations of various types of endothelial cells can also be made using the antibodies to endothelial markers of the invention. The antibodies can be used to purify cell populations according to any technique known in the art, including but not limited to fluorescence activated cell sorting. Such techniques permit the isolation of populations which are at least 50, 60, 70, 80, 90, 92, 94, 95, 96, 97, 98, and even 99 % the type of endothelial cell desired, whether normal, tumor, or pan-endothelial. Antibodies can be used to both positively select and negatively select such populations. Preferably at least 1, 5, 10, 15, 20, or 25 of the appropriate markers are expressed by the endothelial cell population.

- [113] Populations of endothelial cells made as described herein, can be used for screening drugs to identify those suitable for inhibiting the growth of tumors by virtue of inhibiting the growth of the tumor vasculature.
- [114] Populations of endothelial cells made as described herein, can be used for screening candidate drugs to identify those suitable for modulating angiogenesis, such as for inhibiting the growth of tumors by virtue of inhibiting the growth of endothelial cells, such as inhibiting the growth of the tumor or other undesired vasculature, or alternatively, to promote the growth of endothelial cells and thus stimulate the growth of new or additional large vessel or microvasculature.
- [115] Inhibiting the growth of endothelial cells means either regression of vasculature which is already present, or the slowing or the absence of the development of new vascularization in a treated system as compared with a control system. By stimulating the growth of endothelial cells, one can influence development of new (neovascularization) or additional vasculature development (revascularization). A variety of model screen systems are available in which to test the angiogenic and/or anti-angiogenic properties of a given candidate drug. Typical tests involve assays measuring the endothelial cell response, such as proliferation, migration, differentiation and/or intracellular interaction of a given candidate drug. By such tests, one can study the signals and effects of the test stimuli. Some common screens involve measurement of the inhibition of heparanase, endothelial tube formation on Matrigel, scratch induced motility of endothelial cells, platelet-derived growth factor driven proliferation of vascular smooth muscle cells, and the rat aortic ring assay (which provides an advantage of capillary formation rather than just one cell type).
- [116] Drugs can be screened for the ability to mimic or modulate, inhibit or stimulate, growth of tumor endothelium cells and/or normal endothelial cells. Drugs can be screened for the ability to inhibit tumor endothelium growth but not normal endothelium growth or survival. Similarly, human cell

populations, such as normal endothelium populations or tumor endothelial cell populations, can be contacted with test substances and the expression of tumor endothelial markers and/or normal endothelial markers determined. Test substances which decrease the expression of tumor endothelial markers (TEMs) are candidates for inhibiting angiogenesis and the growth of tumors. Conversely, markers which are only expressed in normal endothelium but not in tumor endothelium (NEMs) can be monitored. Test substances which increase the expression of such NEMs in tumor endothelium and other human cells can be identified as candidate antitumor or anti-angiogenic drugs. In cases where the activity of a TEM or NEM is known, agents can be screened for their ability to decrease or increase the activity.

[117] For those tumor endothelial markers identified as containing transmembrane regions, it is desirable to identify drug candidates capable of binding to the TEM receptors found at the cell surface. For some applications, the identification of drug candidates capable of blocking the TEM receptor from its native ligand will be desired. For some applications, the identification of a drug candidate capable of binding to the TEM receptor may be used as a means to deliver a therapeutic or diagnostic agent. For other applications, the identification of drug candidates capable of mimicking the activity of the native ligand will be desired. Thus, by manipulating the binding of a transmembrane TEM:receptor:ligand complex, one may be able to promote or inhibit further development of endothelial cells and hence, vascularization.

[118] For those tumor endothelial markers identified as being secreted proteins, it is desirable to identify drug candidates capable of binding to the secreted TEM protein. For some applications, the identification of drug candidates capable of interfering with the binding of the secreted TEM to its native receptor. For other applications, the identification of drug candidates capable of mimicking the activity of the native receptor will be desired. Thus, by manipulating the binding of the secreted TEM:receptor complex, one may be able to promote or inhibit further development of endothelial cells, and hence, vascularization.

- [119] Expression can be monitored according to any convenient method. Protein or mRNA can be monitored. Any technique known in the art for monitoring specific genes' expression can be used, including but not limited to ELISAs, SAGE, microarray hybridization, Western blots. Changes in expression of a single marker may be used as a criterion for significant effect as a potential pro-angiogenic, anti-angiogenic or anti-tumor agent. However, it also may be desirable to screen for test substances which are able to modulate the expression of at least 5, 10, 15, or 20 of the relevant markers, such as the tumor or normal endothelial markers. Inhibition of TEM protein activity can also be used as a drug screen. Human and mouse TEMs can be used for this purpose.
- [120] Test substances for screening can come from any source. They can be libraries of natural products, combinatorial chemical libraries, biological products made by recombinant libraries, etc. The source of the test substances is not critical to the invention. The present invention provides means for screening compounds and compositions which may previously have been overlooked in other screening schemes. Nucleic acids and the corresponding encoded proteins of the markers of the present invention can be used therapeutically in a variety of modes. NEMs, can be used to restrict, diminish, reduce, or inhibit proliferation of tumor or other abnormal or undesirable vasculature. TEMs can be used to stimulate the growth of vasculature, such as for wound healing or to circumvent a blocked vessel. The nucleic acids and encoded proteins can be administered by any means known in the art. Such methods include, using liposomes, nanospheres, viral vectors, non-viral vectors comprising polycations, etc. Suitable viral vectors include adenovirus, retroviruses, and sindbis virus. Administration modes can be any known in the art, including parenteral, intravenous, intramuscular, intraperitoneal, topical, intranasal, intrarectal, intrabronchial, etc.
- [121] Specific biological antagonists of TEMs can also be used to therapeutic benefit. For example, antibodies, T cells specific for a TEM, antisense to a TEM, and ribozymes specific for a TEM can be used to restrict, inhibit,

reduce, and/or diminish tumor or other abnormal or undesirable vasculature growth. Such antagonists can be administered as is known in the art for these classes of antagonists generally. Anti-angiogenic drugs and agents can be used to inhibit tumor growth, as well as to treat diabetic retinopathy, rheumatoid arthritis, psoriasis, polycystic kidney disease (PKD), and other diseases requiring angiogenesis for their pathologies.

[122] Mouse counterparts to human TEMS can be used in mouse cancer models or in cell lines or *in vitro* to evaluate potential anti-angiogenic or anti-tumor compounds or therapies. Their expression can be monitored as an indication of effect. Mouse TEMs are disclosed in SEQ ID NO: 182-186 and 190-194. Mouse TEMs can be used as antigens for raising antibodies which can be tested in mouse tumor models. Mouse TEMs with transmembrane domains are particularly preferred for this purpose. Mouse TEMs can also be used as vaccines to raise an immunological response in a human to the human ortholog.

[123] The above disclosure generally describes the present invention. All references disclosed herein are expressly incorporated by reference. A more complete understanding can be obtained by reference to the following specific examples which are provided herein for purposes of illustration only, and are not intended to limit the scope of the invention.

EXAMPLE 1

Visualization of vasculature of colorectal cancers

[124] The endothelium of human colorectal cancer was chosen to address the issues of tumor angiogenesis, based on the high incidence, relatively slow growth, and resistance to anti-neoplastic agents of these cancers. While certain less common tumor types, such as glioblastomas, are highly vascularized and are regarded as good targets for anti-angiogenic therapy, the importance of

angiogenesis for the growth of human colorectal cancers and other common solid tumor types is less well documented.

- [125] We began by staining vessels in colorectal cancers using von Willebrand Factor (vWF) as a marker. In each of 6 colorectal tumors, this examination revealed a high density of vessels throughout the tumor parenchyma (Examples in Fig. 1 A and B). Interestingly, these analyses also substantiated the importance of these vessels for tumor growth, as endothelium was often surrounded by a perivascular cuff of viable cells, with a ring of necrotic cells evident at the periphery (Example in Fig. 1A). Although these preliminary studies suggested that colon tumors are angiogenesis-dependent, reliable markers that could distinguish vessels in colon cancers from the vessels in normal colon are currently lacking. One way to determine if such markers exist is by analyzing gene expression profiles in endothelium derived from normal and neoplastic tissue.

EXAMPLE 2

Purification of endothelial cells

- [126] Global systematic analysis of gene expression in tumor and normal endothelium has been hampered by at least three experimental obstacles. First, endothelium is enmeshed in a complex tissue consisting of vessel wall components, stromal cells, and neoplastic cells, requiring highly selective means of purifying ECs for analysis. Second, techniques for defining global gene expression profiles were not available until recently. And third, only a small fraction of the cells within a tumor are endothelial, mandating the development of methods that are suitable for the analysis of global expression profiles from relatively few cells.

- [127] To overcome the first obstacle, we initially attempted to purify ECs from dispersed human colorectal tissue using CD31, an endothelial marker

commonly used for this purpose. This resulted in a substantial enrichment of ECs but also resulted in contamination of the preparations by hematopoietic cells, most likely due to expression of CD31 by macrophages. We therefore developed a new method for purifying ECs from human tissues using P1H12, a recently described marker for ECs. Unlike CD31, P1H12 was specifically expressed on the ECs of both colorectal tumors and normal colorectal mucosa. Moreover, immunofluorescence staining of normal and cancerous colon with a panel of known cell surface endothelial markers (e.g. VE-cadherin, CD31 and CD34) revealed that P1H12 was unique in that it stained all vessels including microvessels (see Fig. 2A and data not shown). In addition to selection with P1H12, it was necessary to optimize the detachment of ECs from their neighbors without destroying their cell surface proteins as well as to employ positive and negative affinity purifications using a cocktail of antibodies (Fig. 2B). The ECs purified from normal colorectal mucosa and colorectal cancers were essentially free of epithelial and hematopoietic cells as judged by RT-PCR (Fig. 2C) and subsequent gene expression analysis (see below).

[128]

EXAMPLE 3

Comparison of tumor and normal endothelial cell expression patterns

[129] To overcome the remaining obstacles, a modification of the Serial Analysis of Gene Expression (SAGE) technique was used. SAGE associates individual mRNA transcripts with 14 base pair tags derived from a specific position near their 3' termini. The abundance of each tag provides a quantitative measure of the transcript level present within the mRNA population studied. SAGE is not dependent on pre-existing databases of expressed genes, and therefore provides an unbiased view of gene expression profiles. This feature is particularly important in the analysis of cells that constitute only a small fraction of the tissue under study, as transcripts from these cells are unlikely to be well represented in extant EST databases. We adapted the SAGE protocol so that it could be used on small numbers of

purified ECs obtained from the procedure outlined in Fig. 2B. A library of ~100,000 tags from the purified ECs of a colorectal cancer, and a similar library from the ECs of normal colonic mucosa from the same patient were generated. These ~193,000 tags corresponded to over 32,500 unique transcripts. Examination of the expression pattern of hematopoietic, epithelial and endothelial markers confirmed the purity of the preparations (Fig. 2D).

EXAMPLE 4

Markers of normal and tumor endothelium

[130] We next sought to identify Pan Endothelial Markers (PEMs), that is, transcripts that were expressed at significantly higher levels in both normal and tumor associated endothelium compared to other tissues. To identify such PEMs, tags expressed at similar levels in both tumor and normal ECs were compared to ~1.8 million tags from a variety of cell lines derived from tumors of non-endothelial origin. This simple comparison identified 93 transcripts that were strikingly EC-specific, i.e. expressed at levels at least 20-fold higher in ECs in vivo compared to non-endothelial cells in culture. The 15 tags corresponding to characterized genes which were most highly and specifically expressed in endothelium are shown in Table 1A. Twelve of these 15 most abundant endothelial transcripts had been previously shown to be preferentially expressed in endothelium, while the other 3 genes had not been associated with endothelium in the past (Table 1A). These data sets also revealed many novel PEMs, which became increasingly prevalent as tag expression levels decreased (Table 1B). For many of the transcripts, their endothelial origin was confirmed by SAGE analysis of ~401,000 transcripts derived from primary cultures of human umbilical vein endothelial cells (HUVEC) and human dermal microvascular endothelial cells (HMVEC) (Table 1 A and B). To further validate the expression of these PEMs in vivo, we developed a highly sensitive non-radioactive in situ hybridization method that allowed the detection of transcripts expressed at relatively low levels in frozen sections of human tissues. Two uncharacterized markers, PEM3 and

PEM6, were chosen for this analysis. In each case, highly specific expression was clearly limited to vascular ECs in both normal and neoplastic tissues (Fig. 3 A and B and data not shown). These data also suggest that ECs maintained in culture do not completely recapitulate expression patterns observed in vivo. For example, Hevin and several other PEM's were expressed at high levels in both tumor and normal ECs in vivo, but few or no transcripts were detected in cultured HUVEC or HMVEC (Table 1). The source of the Hevin transcripts was confirmed to be endothelium by in situ hybridization in normal and malignant colorectal tissue (Fig. 3C).

- [131] Many of the markers reported in Table 1 were expressed at significantly higher levels than previously characterized genes commonly associated with ECs. For example, the top 25 markers were all expressed at greater than 200 copies per cell. In contrast, the receptors for VEGF (VEGFR-1 and VEGFR-2) were expressed at less than 20 copies per cell. Interestingly, VEGFR2 (KDR), which had previously been reported to be up-regulated in vessels during colon cancer progression, was found to be expressed in both normal and neoplastic colorectal tissue (Fig. 3 D and E). The lack of specificity of this gene was in accord with the SAGE data, which indicated that the VEGFR was expressed at 12 copies per cell in both normal and tumor endothelium.

EXAMPLE 5

Tumor *versus* normal endothelium

- [132] We next attempted to identify transcripts that were differentially expressed in endothelium derived from normal or neoplastic tissues. This comparison revealed 33 tags that were preferentially expressed in normal-derived endothelium at levels at least 10-fold higher than in tumor-derived endothelium. Conversely, 46 tags were expressed at 10-fold or higher levels in tumor vessels. Because those transcripts expressed at higher levels in tumor endothelium are most likely to be useful in the future for diagnostic and therapeutic purposes, our subsequent studies focussed on this class. Of the top

25 tags most differentially expressed, 12 tags corresponded to 11 previously identified genes, one with an alternative polyadenylation site (see Table 2). Of these 10 genes, 6 have been recognized as markers associated with angiogenic vessels. The remaining 14 tags corresponded to uncharacterised genes, most of which have only been deposited as ESTs (Table 2).

- [133] To validate the expression patterns of these genes, we chose to focus on 9 Tumor Endothelial Markers (BSC-TEM 1-9; TEM 1, 2, 5, 9, 16, 17, 19, and 22) for which EST sequences but no other information was available (Table 2). These tags were chosen simply because they were among the most differentially expressed on the list and because we were able to obtain suitable probes. In many cases, this required obtaining near full-length sequences through multiple rounds of sequencing and cDNA walking (See accession numbers in Table 2). RT-PCR analysis was then used to evaluate the expression of the corresponding transcripts in purified ECs derived from normal and tumor tissues of two patients different from the one used to construct the SAGE libraries. As shown in Fig. 4 A, the vWF gene, expected to be expressed in both normal and tumor endothelium on the basis of the SAGE data as well as previous studies, was expressed at similar levels in normal and tumor ECs from both patients, but was not expressed in purified tumor epithelial cells. As expected, PEM2 displayed a pattern similar to vWF. In contrast, all 9 TEMs chosen for this analysis were prominently expressed in tumor ECs, but were absent or barely detectable in normal ECs (Table 3 and examples in Fig. 4A). It is important to note that these RT-PCR assays were extremely sensitive indicators of expression, and the absence of detectable transcripts in the normal endothelium, combined with their presence in tumor endothelial RNAs even when diluted 100-fold, provides compelling confirmatory evidence for their differential expression. These results also show that these transcripts were not simply expressed differentially in the ECs of the original patient, but were characteristic of colorectal cancer endothelium in general.

- [134] It could be argued that the results noted above were compromised by the possibility that a small number of non-endothelial cells contaminated the cell populations used for SAGE and RT-PCR analyses, and that these non-endothelial cells were responsible for the striking differences in expression of the noted transcripts. To exclude this possibility, we performed in situ hybridization on normal and neoplastic colon tissue. In every case where transcripts could be detected (BSC-TEM 1, 3, 4, 5, 7, 8, and 9; TEM 1, 5, 9, 17, and 19), they were specifically localized to ECs (Table 3 and examples in Fig. 4 B and C). Although caution must be used when interpreting negative in situ hybridization results, none of the TEMs were expressed in vascular ECs associated with normal colorectal tissue even though vWF and Hevin were clearly expressed (Table 3).

EXAMPLE 6

Tumor endothelium markers are expressed in multiple tumor types

- [135] Were these transcripts specifically expressed in the endothelium within primary colorectal cancers, or were they characteristic of tumor endothelium in general? To address this question, we studied the expression of a representative TEM (BSC-TEM7; TEM 17) in a liver metastasis from a colorectal cancer, a sarcoma, and in primary cancers of the lung, pancreas, breast and brain. As shown in Fig. 4, the transcript was found to be expressed specifically in the endothelium of each of these cancers, whether metastatic (Fig. 4D) or primary (Fig. 4E-I). Analysis of the other six TEMs, (BSC-TEM 1, 3, 4, 5, 7, 8 and 9; TEM 1, 5, 9, 17, and 19) revealed a similar pattern in lung tumors, brain tumors, and metastatic lesions of the liver (see Table 3).

EXAMPLE 7

Tumor endothelium markers are neo-angiogenic

[136] Finally, we asked whether these transcripts were expressed in angiogenic states other than that associated with tumorigenesis. We thus performed in situ hybridizations on corpus luteum tissue as well as healing wounds. Although there were exceptions, we found that these transcripts were generally expressed both in the corpus luteum and in the granulation tissue of healing wounds (Table 3 and example in Fig. 4J). In all tissues studied, expression of the genes was either absent or exclusively confined to the EC compartment.

References and Notes

The disclosure of each reference cited is expressly incorporated herein.

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8. The original EC isolation protocol was the same as that shown in Fig. 2B except that dispersed cells were stained with anti-CD31 antibodies instead of anti-P1H12, and magnetic beads against CD64 and CD14 were not included in the negative selection. After generating 120,000 SAGE tags from these two EC preparations, careful analysis of the SAGE data revealed that, in addition to endothelial-specific markers, several macrophage-specific markers were also present.
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11. In order to reduce the minimum amount of starting material required from ~50 million cells to ~50,000 cells (i.e. ~1000-fold less) we and others (38) have introduced several modifications to the original SAGE protocol. A detailed version of our modified "MicroSAGE" protocol is available from the authors upon request.

12. 96,694 and 96,588 SAGE tags were analyzed from normal and tumor derived ECs, respectively, and represented 50,298 unique tags. A conservative estimate of 32,703 unique transcripts was derived by considering only those tags observed more than once in the current data set or in the 134,000 transcripts previously identified in human transcriptomes (39).

13. To identify endothelial specific transcripts, we normalized the number of tags analyzed in each group to 100,000, and limited our analysis to transcripts that were expressed at levels at least 20-fold higher in ECs than in non-endothelial cell lines in culture and present at fewer than 5 copies per 100,000 transcripts in non-endothelial cell lines and the hematopoietic fraction (~57,000 tags)(41). Non-endothelial cell lines consisted of 1.8x10⁶ tags derived from a total of 14 different cancer cell lines including colon, breast, lung, and pancreatic cancers, as well as one non-transformed keratinocyte cell line, two kidney epithelial cell lines, and normal monocytes. A complete list of PEMs is available at www.sagenet.org/angio/table1.htm.

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26. For non-radioactive in situ hybridization, digoxigenin (DIG)-labelled sense and anti-sense riboprobes were generated through PCR by amplifying 500-600 bp products and incorporating a T7 promoter into the anti-sense primer. In vitro transcription was performed using DIG RNA labelling reagents and T7 RNA polymerase (Roche, Indianapolis, IN). Frozen tissue sections were fixed with 4 % paraformaldehyde, permeabilized with pepsin, and incubated with 200 ng/ml of riboprobe overnight at 55°C. For signal amplification, a horseradish peroxidase (HRP) rabbit anti-DIG antibody (DAKO, Carpinteria, CA) was used to catalyse the deposition of Biotin-Tyramide (from GenPoint kit, DAKO). Further amplification was achieved by adding HRP rabbit anti-biotin (DAKO), biotin-tyramide, and then alkaline-phosphatase (AP) rabbit anti-biotin (DAKO). Signal was detected using the AP substrate Fast Red TR/Naphthol AS-MX (Sigma, St. Louis, MO), and cells were counterstained with hematoxylin unless otherwise indicated. A detailed protocol including the list of primers used to generate the probes can be obtained from the authors upon request.
27. Transcript copies per cell were calculated assuming an average cell contains 300,000 transcripts.
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31. Endothelial-specific transcripts were defined as those expressed at levels at least 5-fold higher in ECs in vivo than in non-endothelial cell lines in culture (13), and present at no more than 5 copies per 100,000 transcripts in non-endothelial cell lines and the hematopoietic cell fraction (41). Transcripts showing statistically different levels of expression ($P < 0.05$) were then identified using Monte Carlo analysis as previously described (40). Transcripts preferentially expressed in normal endothelium were then defined as those expressed at levels at least 10-fold higher in normal endothelium than in tumor endothelium. Conversely, tumor endothelial transcripts were at least 10-fold higher in tumor versus normal endothelium. See www.sagenet.org/angio/table2.htm and www.sagenet.org/angio/table3.htm for a complete list of differentially expressed genes.

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41. Human colon tissues were obtained within ½ hour after surgical removal from patients. Sheets of epithelial cells were peeled away from normal tissues with a glass slide following treatment with 5 mM DDT, then 10 mM EDTA, leaving the lamina propria intact. After a 2h incubation in collagenase at 37 °C, cells were filtered

sequentially through 400 um, 100 um, 50 um and 25 um mesh, and spun through a 30 % pre-formed Percoll gradient to pellet RBCs. Epithelial cells (Epithelial Fraction), which were found to non-specifically bind magnetic beads, were removed using Dynabeads coupled to BerEP4 (DynaL, Lake Success, NY). Subsequently, macrophages and other leukocytes (Hematopoietic Fraction) were removed using a cocktail of beads coupled to anti-CD45, anti-CD14 and anti-CD64 (DynaL). The remaining cells were stained with P1H12 antibody, purified with anti-mouse IgG-coupled magnetic beads, and lysed in mRNA lysis buffer. A detailed protocol can be obtained from the authors upon request.

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Sequence name	SEQ ID NO:
PEM 1	1
PEM 2	2
PEM 3	3
PEM 4	4
PEM 5	5
PEM 6	6
PEM 7	7
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PEM 10	10
PEM 11	11
PEM 12	12
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TEM 7B Protein	189
mTEM 1 Protein	190
mTEM 5 Protein	191
mTEM 7 Protein	192
mTEM 7b Protein	193
mTEM 8 Protein	194
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357	TEM 46 long tag
358	TEM 35 Protein
359	TEM 3 DNA with 5'UTR

CLAIMS

1. An isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of TEM 3 protein as shown in SEQ ID NO: 200.
2. The isolated molecule of claim 1 which is an intact antibody molecule.
3. The isolated molecule of claim 1 which is a single chain variable region (ScFv).
4. The isolated molecule of claim 1 which is a monoclonal antibody.
5. The isolated molecule of claim 1 which is a humanized antibody.
6. The isolated molecule of claim 1 which is a human antibody.
7. The isolated molecule of claim 1 which is bound to a cytotoxic moiety.
8. The isolated molecule of claim 1 which is bound to a therapeutic moiety.
9. The isolated molecule of claim 1 which is bound to a detectable moiety.
10. The isolated molecule of claim 1 which is bound to an anti-tumor agent.
11. A method of inhibiting neoangiogenesis, comprising:
administering to a subject in need thereof an effective amount of an isolated molecule comprising an antibody

variable region which specifically binds to an extracellular domain of TEM 3 protein as shown in SEQ ID NO: 200, whereby neoangiogenesis is inhibited.

12. The method of claim 11 wherein the subject bears a vascularized tumor.

13. The method of claim 11 wherein the subject has polycystic kidney disease.

14. The method of claim 11 wherein the subject has diabetic retinopathy.

15. The method of claim 11 wherein the subject has rheumatoid arthritis.

16. The method of claim 11 wherein the subject has psoriasis.

17. A method of inhibiting tumor growth, comprising:

administering to a human subject bearing a tumor an effective amount of an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of TEM 3 protein as shown in SEQ ID NO: 200, whereby growth of the tumor is inhibited.

18. An isolated molecule comprising an antibody variable region which specifically binds to TEM 3 protein as shown in SEQ ID NO: 200.

19. The isolated molecule of claim 18 which is a single chain variable region (ScFv).

20. The isolated molecule of claim 18 which is a monoclonal antibody.
21. The isolated molecule of claim 18 which is a humanized antibody.
22. The isolated molecule of claim 18 which is a human antibody.
23. The isolated molecule of claim 18 which is bound to a cytotoxic moiety.
24. The isolated molecule of claim 18 which is bound to a therapeutic moiety.
25. The isolated molecule of claim 18 which is bound to a detectable moiety.
26. The isolated molecule of claim 18 which is bound to an anti-tumor agent.
27. The isolated molecule of claim 18 which is an intact antibody molecule.
28. An isolated and purified human transmembrane protein: TEM 3, as shown in SEQ ID NO: 200.
29. An isolated and purified nucleic acid molecule comprising a coding sequence for a transmembrane TEM 3 as shown in SEQ ID NO: 200.
30. The isolated and purified nucleic acid molecule of claim 29 which comprises a coding sequence selected from those shown in SEQ ID NO: 199 and 359.

31. A recombinant host cell which comprises a nucleic acid molecule comprising a coding sequence for a transmembrane TEM 3 as shown in SEQ ID NO: 200.
32. The recombinant host cell of claim 31 which comprises a coding sequence selected from those shown in SEQ ID NO: 199 and 359.
33. A method of inducing an immune response in a mammal, comprising:
administering to the mammal a nucleic acid molecule comprising a coding sequence for a human transmembrane protein TEM 3 as shown in SEQ ID NO: 200, whereby an immune response to the human transmembrane protein is induced in the mammal.
34. The method of claim 33 wherein the coding sequence is shown in SEQ ID NO: 199.
35. A method of inducing an immune response in a mammal, comprising:
administering to the mammal a purified human transmembrane protein TEM 3 as shown in SEQ ID NO: 200, whereby an immune response to the human transmembrane protein is induced in the mammal.
36. A method for identification of a ligand involved in endothelial cell regulation, comprising:
contacting a test compound with an isolated and purified human transmembrane protein TEM 3 as shown in SEQ ID NO: 200;

contacting the isolated and purified human transmembrane protein with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein 3, as shown in SEQ ID NO: 200;

determining binding of the molecule comprising an antibody variable region to the human transmembrane protein, wherein a test compound which diminishes the binding of the molecule comprising an antibody variable region to the human transmembrane protein is identified as a ligand involved in endothelial cell regulation.

37. A method for identification of a ligand involved in endothelial cell regulation, comprising:

contacting a test compound with a cell comprising a human transmembrane protein 3 as shown in SEQ ID NO: 200;

contacting the cell with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein selected from the group consisting of: 3 as shown in SEQ ID NO:200;

determining binding of the molecule comprising an antibody variable region to the cell, wherein a test compound which diminishes the binding of the molecule comprising an antibody variable region to the cell is identified as a ligand involved in endothelial cell regulation.

38. A soluble form of a human transmembrane protein TEM 3 as shown in SEQ ID NO: 200, respectively, wherein the soluble forms lack transmembrane domains.

39. The soluble form of claim 38 wherein the soluble form consists of an extracellular domain of the human transmembrane protein.
40. A method of inhibiting neoangiogenesis in a patient, comprising:
administering to the patient a soluble form of a human transmembrane protein according to claim 38, whereby neoangiogenesis in the patient is inhibited.
41. A method of inhibiting neoangiogenesis in a patient, comprising:
administering to the patient a soluble form of a human transmembrane protein according to claim 39, whereby neoangiogenesis in the patient is inhibited.
42. The method of claim 40 wherein the patient bears a vascularized tumor.
43. The method of claim 41 wherein the patient bears a vascularized tumor.
44. The method of claim 40 wherein the patient has polycystic kidney disease.
45. The method of claim 40 wherein the patient has diabetic retinopathy.
46. The method of claim 40 wherein the patient has rheumatoid arthritis.
47. The method of claim 40 wherein the patient has psoriasis.

48. The method of claim 41 wherein the patient has polycystic kidney disease.

49. The method of claim 41 wherein the patient has diabetic retinopathy.

50. The method of claim 41 wherein the patient has rheumatoid arthritis.

51. The method of claim 41 wherein the patient has psoriasis.

52. A method of identifying regions of neoangiogenesis in a patient, comprising:

administering to a patient a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein 3 as shown in SEQ ID NO: 200, wherein the molecule is bound to a detectable moiety; and

detecting the detectable moiety in the patient, thereby identifying neoangiogenesis.

53. A method of screening for neoangiogenesis in a patient, comprising:

contacting a body fluid collected from the patient with a molecule comprising an antibody variable region which specifically binds to an extracellular domain of a TEM protein 3 as shown in SEQ ID NO: 200, wherein detection of cross-reactive material in the body fluid with the molecule indicates neoangiogenesis in the patient.

54. A method of inducing an immune response to tumor endothelial cells in a patient, comprising:

administering to a patient in need thereof a mouse TEM protein selected from the group consisting of: 1, 2, 3, 9, 13, 17, 19, 22, and 30 as shown in SEQ ID NO: 291, 293, 299, 295, 303, 297, 301, 305, and 307, whereby an immune response to a human TEM protein is induced.

55. A method to identify candidate drugs for treating tumors, comprising:

contacting cells which express TEM 3 gene as shown in SEQ ID NO: 199 with a test compound;

determining expression of said one or more TEM genes by hybridization of mRNA of said cells to a nucleic acid probe which is complementary to said mRNA; and

identifying a test compound as a candidate drug for treating tumors if it decreases expression of said one or more TEM genes.

56. The method of claim 55 wherein the cells are endothelial cells.

57. The method of claim 55 wherein the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs.

58. A method to identify candidate drugs for treating tumors, comprising:

contacting cells which express TEM 3 protein as shown in SEQ ID NO: 200, with a test compound;

determining amount of said one or more TEM proteins in said cells; and

identifying a test compound as a candidate drug for treating tumors if it decreases the amount of one more TEM proteins in said cells.

59. The method of claim 58 wherein the cells are endothelial cells.

60. The method of claim 58 wherein the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs.

61. A method to identify candidate drugs for treating tumors, comprising:

contacting cells which express TEM 3 protein as shown in SEQ ID NO: 200 with a test compound;

determining activity of said one or more TEM proteins in said cells; and

identifying a test compound as a candidate drug for treating tumors if it decreases the activity of of one more TEM proteins in said cells.

62. The method of claim 61 wherein the cells are endothelial cells.

63. The method of claim 61 wherein the cells are recombinant host cells which are transfected with an expression construct which encodes said one or more TEMs.

64. A method to identify candidate drugs for treating patients bearing tumors, comprising:

contacting a test compound with recombinant host cells which are transfected with an expression construct which encodes TEM 3 proteins as shown in SEQ ID NO: 200;

determining proliferation of said cells; and

identifying a test compound which inhibits proliferation of said cells as a candidate drug for treating patients' bearing tumors.

65. A method for identification of a ligand involved in endothelial cell regulation, comprising:

contacting a test compound with a human transmembrane protein TEM 3 as shown in SEQ ID NO: 200;

determining binding of a test compound to the human transmembrane protein, wherein a test compound which binds to the protein is identified as a ligand involved in endothelial cell regulation.

66. A method of inducing an immune response in a mammal, comprising:

administering to the mammal a cell which expresses a transmembrane protein TEM 3 as shown in SEQ ID NO: 200, wherein the cell is a recombinant cell which comprises a vector encoding said transmembrane protein, or the cell is a fusion of a dendritic cell and a tumor endothelium cell, whereby an immune response to the human transmembrane protein is induced in the mammal.

67. A method of inhibiting neoangiogenesis, comprising:

administering to a subject in need thereof an effective amount of an isolated molecule comprising an antibody variable region which specifically binds to an extracellular domain of a mouse TEM protein selected from the group

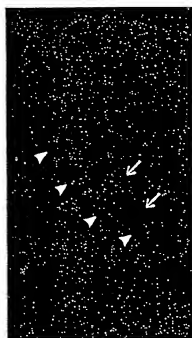
consisting of: 1, 2, 3, 9, 17, and 19, as shown in SEQ ID NO:
291, 293, 299, 295, 297, and 301, respectively, whereby
neoangiogenesis is inhibited.

68. The method of claim 87 wherein the subject is a mouse.
69. The method of claim 87 wherein the subject bears a
vascularized tumor.
70. The method of claim 87 wherein the subject has polycystic
kidney disease.
71. The method of claim 87 wherein the subject has diabetic
retinopathy.
72. The method of claim 87 wherein the subject has rheumatoid
arthritis.
73. The method of claim 87 wherein the subject has psoriasis.

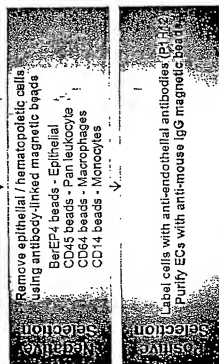
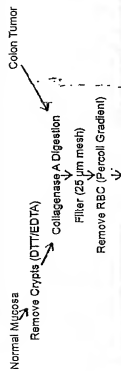


Figure 1

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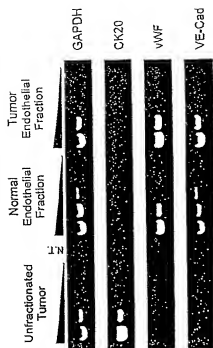


B



Lyse cells, isolate RNA

C



D

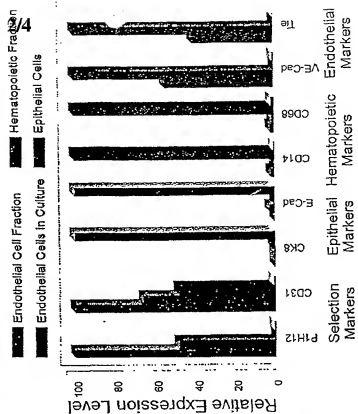


Figure 2

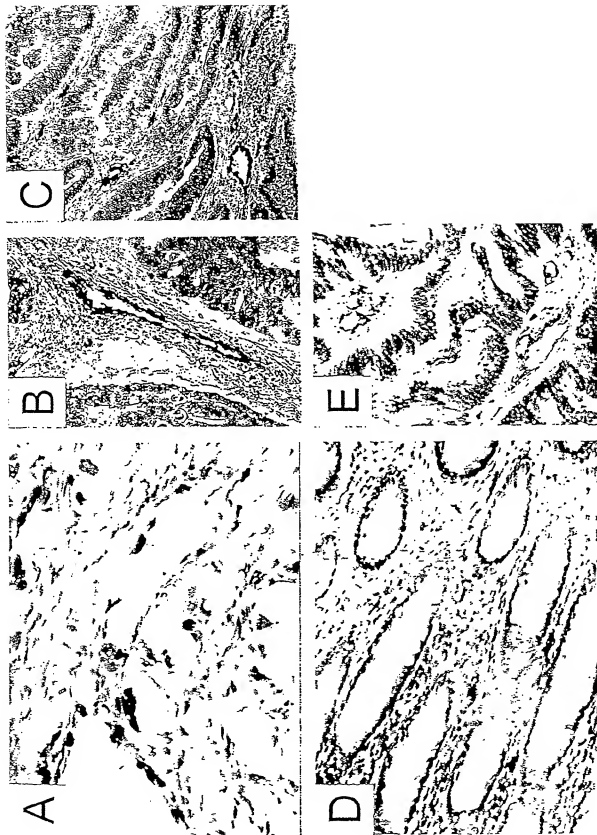


Figure 3

A

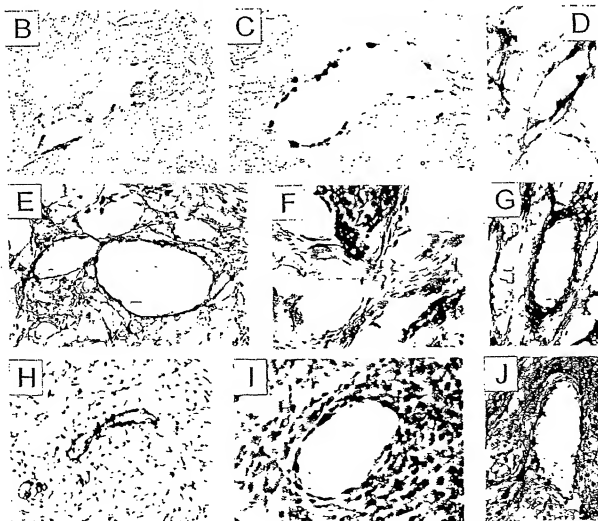
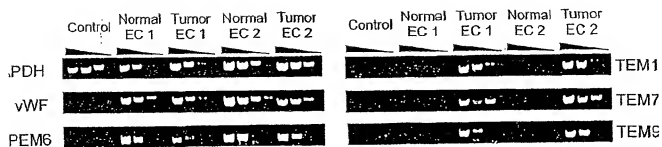


Figure 4

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Vogelstein, Bert
Kinzler, Kenneth

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Gly	Cys	Glu	His	Glu	Cys	Val	Glu	Glu	Val	Asp	Gly	His	Val	Ser	Cys
			245					250					255		
Arg	Cys	Thr	Glu	Gly	Phe	Arg	Leu	Ala	Ala	Asp	Gly	Arg	Ser	Cys	Glu
			260				265						270		
Asp	Pro	Cys	Ala	Gln	Ala	Pro	Cys	Glu	Gln	Gln	Cys	Glu	Pro	Gly	Gly
			275			280						285			
Pro	Gln	Gly	Tyr	Ser	Cys	His	Cys	Arg	Leu	Gly	Phe	Arg	Pro	Ala	Glu
			290			295						300			
Asp	Asp	Pro	His	Arg	Cys	Val	Asp	Thr	Asp	Glu	Cys	Gln	Ile	Ala	Gly

305 310 315 320
 Val Cys Gln Gln Met Cys Val Asn Tyr Val Gly Gly Phe Glu Cys Tyr
 325 330 335
 Cys Ser Glu Gly His Glu Leu Glu Ala Asp Gly Ile Ser Cys Ser Pro
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 Ala Gly Ala Met Gly Ala Gln Ala Ser Gln Asp Leu Gly Asp Glu Leu
 355 360 365
 Leu Asp Asp Gly Glu Asp Glu Asp Glu Asp Glu Ala Trp Lys Ala
 370 375 380
 Phe Asn Gly Gly Trp Thr Glu Met Pro Gly Ile Leu Trp Met Glu Pro
 385 390 395 400
 Thr Gln Pro Pro Asp Phe Ala Leu Ala Tyr Arg Pro Ser Phe Pro Glu
 405 410 415
 Asp Arg Glu Pro Gln Ile Pro Tyr Pro Glu Pro Thr Trp Pro Pro Pro
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 Leu Ser Ala Pro Arg Val Pro Tyr His Ser Ser Val Leu Ser Val Thr
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 Arg Pro Val Val Val Ser Ala Thr His Pro Thr Leu Pro Ser Ala His
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 Gln Pro Pro Val Ile Pro Ala Thr His Pro Ala Leu Ser Arg Asp His
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 Leu Gly Ala Gln Leu Pro Pro Gln Ala Pro Asp Ala Leu Val Leu Arg
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 Thr Gln Ala Thr Gln Leu Pro Ile Ile Pro Thr Ala Gln Pro Ser Leu
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 595 600 605
 Pro Ala Ala Thr Gln Pro Ala Ala Leu Pro Thr Leu Leu Pro Ser Gln
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 Ser Pro Thr Asn Gln Thr Ser Pro Ile Ser Pro Thr His Pro His Ser
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 Lys Ala Pro Gln Ile Pro Arg Glu Asp Gly Pro Ser Pro Lys Leu Ala
 645 650 655
 Leu Trp Leu Pro Ser Pro Ala Pro Thr Ala Ala Pro Thr Ala Leu Gly
 660 665 670
 Glu Ala Gly Leu Ala Glu His Ser Gln Arg Asp Asp Arg Trp Leu Leu
 675 680 685
 Val Ala Leu Leu Val Pro Thr Cys Val Phe Leu Val Val Leu Leu Ala
 690 695 700
 Leu Gly Ile Val Tyr Cys Thr Arg Cys Gly Pro His Ala Pro Asn Lys
 705 710 715 720
 Arg Ile Thr Asp Cys Tyr Arg Trp Val Ile His Ala Gly Ser Lys Ser
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 Pro Thr Glu Pro Met Pro Pro Arg Gly Ser Leu Thr Gly Val Gln Thr
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 Cys Arg Thr Ser Val
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<211> 278

<212> FRT

<213> Homo sapiens

<400> 178

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 Arg Met Val Val Leu Gly Ala Ser Arg Val Gly Lys Ser Ser Ile Val
 35 40 45
 Ser Arg Phe Leu Asn Gly Arg Phe Glu Asp Gln Tyr Thr Pro Thr Ile
 50 55 60
 Glu Asp Phe His Arg Lys Val Tyr Asn Ile Arg Gly Asp Met Tyr Gln
 65 70 75 80
 Leu Asp Ile Leu Asp Thr Ser Gly Asn His Pro Phe Pro Ala Met Arg
 85 90 95
 Arg Leu Ser Ile Leu Thr Gly Asp Val Phe Ile Leu Val Phe Ser Leu
 100 105 110
 Asp Asn Arg Glu Ser Phe Asp Glu Val Lys Arg Leu Gln Lys Gln Ile
 115 120 125
 Leu Glu Val Lys Ser Cys Leu Lys Asn Lys Thr Lys Glu Ala Ala Glu
 130 135 140
 Leu Pro Met Val Ile Cys Gly Asn Lys Asn Asp His Gly Glu Leu Cys
 145 150 155 160
 Arg Gln Val Pro Thr Thr Glu Ala Glu Leu Leu Val Ser Gly Asp Glu
 165 170 175
 Asn Cys Ala Tyr Phe Glu Val Ser Ala Lys Lys Asn Thr Asn Val Asp
 180 185 190
 Glu Met Phe Tyr Val Leu Phe Ser Met Ala Lys Leu Pro His Glu Met
 195 200 205
 Ser Pro Ala Leu His Arg Lys Ile Ser Val Gln Tyr Gly Asp Ala Phe
 210 215 220
 His Pro Arg Pro Phe Cys Met Arg Arg Val Lys Glu Met Asp Ala Tyr
 225 230 235 240
 Gly Met Val Ser Pro Phe Ala Arg Arg Pro Ser Val Asn Ser Asp Leu
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 Asp Lys Cys Thr Ile Gln
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<212> PRT

<213> Homo sapiens

<400> 179

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 35 40 45
 Arg Glu Ser Pro Gly His Val Ser Glu Pro Asp Arg Thr Gln Leu Ser
 50 55 60
 Gln Asp Leu Gly Gly Thr Leu Ala Met Asp Thr Leu Pro Asp Asn
 65 70 75 80
 Arg Thr Arg Val Val Glu Asp Asn His Ser Tyr Tyr Val Ser Arg Leu
 85 90 95
 Tyr Gly Pro Ser Glu Pro His Ser Arg Glu Leu Trp Val Asp Val Ala
 100 105 110
 Glu Ala Asn Arg Ser Gln Val Lys Ile His Thr Ile Leu Ser Asn Thr
 115 120 125
 His Arg Gln Ala Ser Arg Val Val Leu Ser Phe Asp Phe Pro Phe Tyr
 130 135 140
 Gly His Pro Leu Arg Gln Ile Thr Ile Ala Thr Gly Gly Phe Ile Phe
 145 150 155 160
 Met Gly Asp Val Ile His Arg Met Leu Thr Ala Thr Gln Tyr Val Ala
 165 170 175
 Pro Leu Met Ala Asn Phe Asn Pro Gly Tyr Ser Asp Asn Ser Thr Val
 180 185 190
 Val Tyr Phe Asp Asn Gly Thr Val Phe Val Val Gln Trp Asp His Val

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 Tyr Leu Gln Gly Trp Glu Asp Lys Gly Ser Phe Thr Phe Gln Ala Ala
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 Leu His His Asp Gly Arg Ile Val Phe Ala Tyr Lys Glu Ile Pro Met
 225
 Ser Val Pro Glu Ile Ser Ser Ser Gln His Pro Val Lys Thr Gly Leu
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 Ser Asp Ala Phe Met Ile Leu Asn Pro Ser Pro Asp Val Pro Glu Ser
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 Arg Arg Arg Ser Ile Phe Glu Tyr His Arg Ile Glu Leu Asp Pro Ser
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 Lys Val Thr Ser Met Ser Ala Val Glu Phe Thr Pro Leu Pro Thr Cys
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 Leu Gln His Arg Ser Cys Asp Ala Cys Met Ser Ser Asp Leu Thr Phe
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 Asn Cys Ser Trp Cys His Val Leu Gln Arg Cys Ser Ser Gly Phe Asp
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 Arg Tyr Arg Gln Glu Trp Asp Gly Thr Met Gly Cys Ala Gln Glu Ala
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 Glu Gly Gln Asp Val Arg Gly Leu Pro Gly Met Arg Thr Thr Thr Ser
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 Ala Ser Pro Asp Thr Ser Phe Ser Pro Tyr Asp Gly Asp Leu Thr Thr
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 Thr Ser Ser Ser Leu Phe Ile Asp Ser Leu Thr Thr Glu Asp Asp Thr
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 Lys Leu Asn Pro Tyr Ala Gly Gly Asp Gly Leu Gln Asn Asn Leu Ser
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 Pro Lys Thr Lys Gly Thr Pro Val His Leu Gly Thr Ile Val Gly Ile
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 Val Leu Ala Val Leu Leu Val Ala Ala Ile Ile Leu Ala Gly Ile Tyr
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 Ser Thr Tyr Ala Glu Val Glu Pro Ser Gly His Glu Lys Glu Gly Phe
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 Met Glu Ala Glu Gln Cys Met Arg Gly Glu Leu Trp Leu Leu Val Leu
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 Val Leu Arg Glu Ala Ala Arg Ala Leu Ser Pro Gln Pro Gly Ala Gly
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 His Asp Glu Gly Pro Gly Ser Gly Trp Ala Ala Lys Gly Thr Val Arg
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 Gly Trp Asn Arg Arg Ala Arg Glu Ser Pro Gly His Val Ser Glu Pro
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 Asp Arg Thr Gln Leu Ser Gln Asp Leu Gly Gly Thr Leu Ala Met
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 Asp Thr Leu Pro Asp Asn Arg Thr Arg Val Val Glu Asp Asn His Ser
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 Tyr Tyr Val Ser Arg Leu Tyr Gly Pro Ser Glu Pro His Ser Arg Glu
 595
 Leu Trp Val Asp Val Ala Glu Ala Asn Arg Ser Gln Val Lys Ile His
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 Thr Ile Leu Ser Asn Thr His Arg Gln Ala Ser Arg Val Val Leu Ser
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 Phe Asp Phe Pro Phe Tyr Gly His Pro Leu Arg Gln Ile Thr Ile Ala
 645
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 Ala Thr Gln Tyr Val Ala Pro Leu Met Ala Asn Phe Asn Pro Gly Tyr
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 Ser Asp Asn Ser Thr Val Val Tyr Phe Asp Asn Gly Thr Val Phe Val
 690
 Val Gln Trp Asp His Val Tyr Leu Gln Gly Trp Glu Asp Lys Gly Ser
 705
 Phe Thr Phe Gln Ala Ala Leu His His Asp Gly Arg Ile Val Phe Ala
 710
 715
 720
 725

Tyr	Lys	Glu	Ile	725	Pro	Met	Ser	Val	Pro	730	Glu	Ile	Ser	Ser	Ser	Ser	Gln	His
Pro	Val	Lys	Thr	740	Leu	Ser	Asp	Ala	Phe	745	Met	Ile	Leu	Asn	Pro	Ser		
Pro	Asp	Val	Pro	755	Glu	Ser	Arg	Arg	Ser	760	Ile	Phe	Glu	Tyr	His	Arg		
Ile	Glu	Leu	Asp	770	Pro	Ser	Lys	Val	Thr	775	Ser	Met	Ser	Ala	Val	Glu	Phe	800
Thr	Pro	Leu	Pro	785	Thr	Cys	Leu	Gln	His	810	Arg	Ser	Cys	Asp	Ala	Cys	Met	
Ser	Ser	Asp	Leu	805	Thr	Phe	Asn	Cys	Ser	825	Tyr	Cys	His	Val	Leu	Gln	Arg	
Cys	Ser	Ser	Gly	835	Phe	Asp	Arg	Tyr	Arg	840	Gln	Glu	Trp	Met	Asp	Tyr	Gly	
Cys	Ala	Gln	Glu	850	Ala	Glu	Gly	Arg	Met	855	Cys	Glu	Asp	Phe	Gln	Asp	Gly	
Asp	His	Asp	Ser	865	Ala	Ser	Pro	Asp	Thr	870	Ser	Phe	Ser	Pro	Tyr	Asp	Gly	
Asp	Leu	Thr	Thr	885	Thr	Ser	Ser	Ser	Leu	890	Phe	Ile	Asp	Ser	Leu	Thr	Thr	
Glu	Asp	Asp	Thr	900	Lys	Leu	Asn	Pro	Tyr	905	Ala	Gly	Gly	Asp	Gly	Leu	Gln	
Asn	Asn	Leu	Ser	915	Pro	Lys	Thr	Lys	Gly	920	Thr	Pro	Val	His	Leu	Gly	Thr	
Ile	Val	Gly	Ile	930	Val	Leu	Ala	Val	Leu	935	Leu	Val	Ala	Ala	Ile	Ile	Leu	
Ala	Gly	Ile	Tyr	945	Ile	Asn	Gly	His	Pro	950	Thr	Ser	Asn	Ala	Ala	Leu	Phe	
Phe	Ile	Glu	Arg	965	Arg	Pro	His	His	Trp	970	Pro	Ala	Met	Lys	Phe	Arg	Ser	
His	Pro	Asp	His	980	Ser	Thr	Tyr	Ala	Glu	985	Val	Glu	Pro	Ser	Gly	His	Glu	
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<211> 5680

<212> DNA

<213> Homo sapiens

<400> 180

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<212> DNA

<213> Mus musculus

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<211> 564

<212> PRT

<213> Homo sapiens

<400> 187

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Phe	Val	Glu	Gln	Leu	Ala	His	Lys	Phe	Ile	Ser	Pro	Gln	Leu	Arg	Met
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Leu	Pro	Gly	Gly	Asp	Thr	Tyr	Met	His	Glu	Gly	Phe	Glu	Arg	Ala	Ser
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 Ile Ile Ala Leu Thr Asp Gly Glu Leu His Glu Asp Leu Phe Phe Tyr
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 Ser Glu Arg Glu Ala Asn Arg Ser Arg Asp Leu Gly Ala Ile Val Tyr
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 Cys Val Gly Val Lys Asp Phe Asn Glu Thr Gln Leu Ala Arg Ile Ala
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 Asp Ser Lys Asp His Val Phe Pro Val Asn Asp Gly Phe Gln Ala Leu
 195 200 205
 Gln Gly Ile Ile His Ser Ile Leu Lys Lys Ser Cys Ile Glu Ile Leu
 210 215 220
 Ala Ala Glu Pro Ser Thr Ile Cys Ala Gly Glu Ser Phe Gln Val Val
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 Cys Ser Phe Lys Ile Asn Asp Ser Val Thr Leu Asn Glu Lys Pro Phe
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 Ser Val Glu Asp Thr Tyr Leu Leu Cys Pro Ala Pro Ile Leu Lys Glu
 275 280 285
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 305 310 315 320
 Ser Ile Leu Ala Ile Ala Leu Leu Ile Leu Phe Leu Leu Leu Ala Leu
 325 330 335
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<210> 188

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<212> PRT

<213> Homo sapiens

<400> 188

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His	Pro	Phe	Gln	Cys	Ser	Ala	Ser	Tyr	Leu	Gly	Asn	Asp	Thr	Arg	Ile
Arg	Trp	Tyr	His	Asn	Arg	Ala	Pro	Val	Glu	Gly	Asp	Glu	Gln	Ala	Gly
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Glu	Leu	Thr	Leu	Ser	His	Ile	Gly	Val	Trp	Ala	Ser	Gly	Glu	Trp	Glu
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Val	Val	Leu	Glu	Thr	Ser	Ala	Ser	Tyr	Cys	Pro	Ala	Glu	Arg	Val	Ala
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Glu	Arg	Ile	Gly	Gly	Ala	Ala	Leu	Ser	Pro	His	Ala	Gln	His	Ile	Ser
Val	Asn	Ala	Arg	Asn	Val	Ala	Leu	Glu	Ala	Tyr	Leu	Ile	Lys	Pro	His
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 Ser Gln Val Cys Glu Ala Gly Ala Ala Ala Gly Gly Glu Gly Glu Pro
 1140 1145 1150
 Glu Pro Ala Gly Thr Arg Gly Asn Leu Ala His Arg His Pro Asn Asn
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 Asn Ser Pro Thr Asp Ser Tyr Leu Gly Ser Ser Arg Asn Ser Pro Gly
 1220 1225 1230
 Ala Gly Leu Gln Leu Glu Gly Glu Pro Met Leu Thr Pro Ser Glu Gly
 1235 1240 1245
 Ser Asp Thr Ser Ala Ala Pro Leu Ser Glu Ala Gly Arg Ala Gly Gln
 1250 1255 1260
 Arg Arg Ser Ala Ser Arg Asp Ser Leu Lys Gly Gly Ala Leu Glu
 1265 1270 1275 1280
 Lys Glu Ser His Arg Arg Ser Tyr Pro Leu Asn Ala Ala Ser Leu Asn
 1285 1290 1295
 Gly Ala Pro Lys Gly Gly Lys Tyr Asp Asp Val Thr Leu Met Gly Ala
 1300 1305 1310
 Glu Val Ala Ser Gly Gly Cys Met Lys Thr Gly Leu Trp Lys Ser Glu
 1315 1320 1325
 Thr Thr Val
 1330

<210> 189

<211> 529

<212> FRT

<213> Homo sapiens

<400> 189

Met Ala Arg Phe Pro Lys Ala Asp Leu Ala Ala Ala Gly Val Met Leu
 1 5 10 15
 Leu Cys His Phe Phe Thr Asp Gln Phe Gln Phe Ala Asp Gly Lys Pro
 20 25 30
 Gly Asp Gln Ile Leu Asp Trp Gln Tyr Gly Val Thr Gln Ala Phe Pro
 35 40 45
 His Thr Glu Glu Glu Val Glu Val Asp Ser His Ala Tyr Ser His Arg
 50 55 60
 Trp Lys Arg Asn Leu Asp Phe Leu Lys Ala Val Asp Thr Asn Arg Ala
 65 70 75 80
 Ser Val Gly Gln Asp Ser Pro Glu Pro Arg Ser Phe Thr Asp Leu Leu
 85 90 95
 Leu Asp Asp Gly Gln Asp Asn Asn Thr Gln Ile Glu Glu Asp Thr Asp
 100 105 110
 His Asn Tyr Tyr Ile Ser Arg Ile Tyr Gly Pro Ser Asp Ser Ala Ser
 115 120 125
 Arg Asp Leu Trp Val Asn Ile Asp Gln Met Glu Lys Asp Lys Val Lys
 130 135 140
 Ile His Gly Ile Leu Ser Asn Thr His Arg Gln Ala Ala Arg Val Asn
 145 150 155
 Leu Ser Phe Asp Phe Pro Phe Tyr Gly His Phe Leu Arg Glu Ile Thr
 165 170 175
 Val Ala Thr Gly Gly Phe Ile Tyr Thr Gly Glu Val Val His Arg Met
 180 185 190
 Leu Thr Ala Thr Gln Tyr Ile Ala Pro Leu Met Ala Asn Phe Asp Pro
 195 200 205

Ser Val Ser Arg Asn Ser Thr Val Arg Tyr Phe Asp Asn Gly Thr Ala
 210 215 220
 Leu Val Val Gln Trp Asp His Val His Leu Gln Asp Asn Tyr Asn Leu
 225 230 235 240
 Gly Ser Phe Thr Phe Gln Ala Thr Leu Leu Met Asp Gly Arg Ile Ile
 245 250 255
 Phe Gly Tyr Lys Glu Ile Pro Val Leu Val Thr Gln Ile Ser Ser Thr
 260 265 270
 Asn His Pro Val Lys Val Gly Leu Ser Asp Ala Phe Val Val Val His
 275 280
 Arg Ile Gln Gln Ile Pro Asn Val Arg Arg Arg Thr Ile Tyr Glu Tyr
 290 295 300
 His Arg Val Glu Leu Gln Met Ser Lys Ile Thr Asn Ile Ser Ala Val
 305 310 315 320
 Glu Met Thr Pro Leu Pro Thr Cys Leu Gln Phe Asn Arg Cys Gly Pro
 325 330 335
 Cys Val Ser Ser Gln Ile Gly Phe Asn Cys Ser Trp Cys Ser Lys Leu
 340 345 350
 Gln Arg Cys Ser Ser Gly Phe Asp Arg His Arg Gln Asp Trp Val Asp
 355 360 365
 Ser Gly Cys Pro Glu Glu Ser Lys Glu Lys Met Cys Glu Asn Thr Glu
 370 375 380
 Pro Val Glu Thr Ser Ser Arg Thr Thr Thr Thr Ile Gly Ala Thr Thr
 385 390 395 400
 Thr Gln Phe Arg Val Leu Thr Thr Thr Arg Arg Ala Val Thr Ser Gln
 405 410 415
 Phe Pro Thr Ser Leu Pro Thr Glu Asp Asp Thr Lys Ile Ala Leu His
 420 425 430
 Leu Lys Asp Asn Gly Ala Ser Thr Asp Asp Ser Ala Ala Glu Lys Lys
 435 440 445
 Gly Gly Thr Leu His Ala Gly Leu Ile Val Gly Ile Leu Ile Leu Val
 450 455 460
 Leu Ile Val Ala Thr Ala Ile Leu Val Thr Val Tyr Met Tyr His His
 465 470 475 480
 Pro Thr Ser Ala Ala Ser Ile Phe Phe Ile Glu Arg Arg Pro Ser Arg
 485 490 495
 Trp Pro Ala Met Lys Phe Arg Arg Gly Ser Gly His Pro Ala Tyr Ala
 500 505 510
 Glu Val Glu Pro Val Gly Glu Lys Glu Gly Phe Ile Val Ser Glu Gln
 515 520 525
 Cys

<210> 190

<211> 765

<212> FRT

<213> Mus musculus

<400> 190

Met Leu Leu Arg Leu Leu Ala Trp Val Ala Ala Val Pro Ala Leu
 1 5 10 15
 Gly Gln Val Pro Trp Thr Pro Glu Pro Arg Ala Ala Cys Gly Pro Ser
 20 25 30
 Ser Cys Tyr Ala Leu Phe Pro Arg Arg Arg Thr Phe Leu Glu Ala Trp
 35 40 45
 Arg Ala Cys Arg Glu Leu Gly Gly Asn Leu Ala Thr Pro Arg Thr Pro
 50 55 60
 Glu Glu Ala Gln Arg Val Asp Ser Leu Val Gly Val Gly Pro Ala Asn
 65 70 75 80
 Gly Leu Leu Trp Ile Gly Leu Gln Arg Gln Ala Arg Gln Cys Gln Pro
 85 90 95
 Gln Arg Pro Leu Arg Gly Phe Ile Trp Thr Thr Gly Asp Gln Asp Thr
 100 105 110
 Ala Phe Thr Asn Trp Ala Gln Pro Ala Thr Glu Gly Pro Cys Pro Ala
 115 120 125
 Gln Arg Cys Ala Ala Leu Glu Ala Ser Gly Glu His Arg Trp Leu Glu

		660						665						670							
Thr	Ala	Ala	Pro	Thr	Ala	Leu	Ala	Glu	Ser	Gly	Leu	Ala	Gly	Gln	Ser						
		675												685							
Gln	Arg	Asp	Asp	Arg	Trp	Leu	Leu	Val	Ala	Leu	Leu	Val	Pro	Thr	Cys						
		690				695						700									
Val	Phe	Leu	Val	Val	Leu	Leu	Ala	Leu	Gly	Ile	Val	Tyr	Cys	Thr	Arg						
705					710					715					720						
Cys	Gly	Ser	His	Ala	Pro	Asn	Lys	Arg	Ile	Thr	Asp	Cys	Tyr	Arg	Trp						
				725					730					735							
Val	Thr	His	Ala	Gly	Asn	Lys	Ser	Ser	Thr	Glu	Pro	Met	Pro	Pro	Arg						
			740					745					750								
Gly	Ser	Leu	Thr	Gly	Val	Gln	Thr	Cys	Arg	Thr	Ser	Val									
		755				760						765									

<210> 191

<211> 1329

<212> PRT

<213> Mus musculus

<400> 191

Met	Pro	Val	Pro	5	Ala	Arg	Leu	Leu	Leu	Pro	Leu	Leu	Pro	Cys
1	Leu	Leu	Leu	10	Pro	Gly	Thr	Arg	Gly	Ala	Pro	Gly	Cys	Pro
Leu	Leu	Leu	Leu	20	Ala	Pro	Gly	Thr	Arg	Gly	Ala	Pro	Gly	Cys
Pro	Ile	Arg	Gly	35	Cys	Lys	Cys	Ser	Gly	Glu	Arg	Pro	Lys	Gly
Gly	Gly	Ala	His	50	Asn	Pro	Ala	Arg	Arg	Arg	Val	Val	Cys	Gly
Asp	Leu	Pro	Glu	65	Pro	Pro	Asp	Pro	Gly	Leu	Leu	Pro	Asn	Gly
Thr	Leu	Leu	Leu	85	Ser	Asn	Asn	Lys	Ile	Thr	Gly	Leu	Arg	Asn
Phe	Leu	Gly	Leu	100	Ser	Leu	Asn	Glu	Lys	Leu	Asp	Leu	Arg	Ser
Ile	Ser	Thr	Val	115	Gln	Pro	Gly	Ala	Phe	Leu	Gly	Leu	Gly	Glu
Arg	Leu	Asp	Leu	130	Ser	Asn	Asn	Arg	Ile	Gly	Cys	Leu	Thr	Ser
Phe	Gln	Gly	Leu	145	Pro	Arg	Leu	Leu	Arg	Leu	Asn	Ile	Ser	Gly
Tyr	Ser	Ser	Leu	165	Gln	Pro	Gly	Val	Phe	Asp	Gly	Leu	Pro	Ala
Ile	Val	Asp	Phe	180	Gly	Thr	Glu	Phe	Leu	Thr	Cys	Asp	Cys	Arg
Trp	Leu	Leu	Pro	195	Trp	Ala	Arg	Asn	His	Ser	Leu	Gln	Leu	Ser
Thr	Leu	Cys	Ala	210	Tyr	Pro	Ser	Ala	Leu	His	Ala	His	Ala	Leu
Leu	Gln	Glu	Ser	225	Gln	Leu	Arg	Cys	Glu	Gly	Ala	Leu	Glu	Leu
His	Tyr	Leu	Ile	245	Pro	Ser	Leu	Arg	Gln	Val	Val	Phe	Gln	Gly
Leu	Pro	Phe	Gln	260	Cys	Ser	Ala	Ser	Tyr	Leu	Gly	Asn	Asp	Thr
His	Trp	Tyr	His	275	Asn	Gly	Ala	Pro	Met	Glu	Ser	Asp	Glu	Gln
Ile	Val	Leu	Ala	290	Glu	Asn	Leu	Ile	His	Asp	Cys	Thr	Phe	Ile
Glu	Ser	Thr	Leu	305	Ser	His	Ile	Gly	Val	Trp	Ala	Ser	Gly	Glu
Cys	Ser	Val	Ser	325	Thr	Val	Gln	Gly	Asn	Thr	Ser	Lys	Lys	Val
Val	Val	Leu	Glu	340	Thr	Ser	Ala	Ser	Tyr	Cys	Pro	Ala	Glu	Arg
Asn	Asn	Arg	Gly	355	Asp	Phe	Arg	Trp	Pro	Arg	Thr	Leu	Ala	Gly

Ala Tyr Gln Ser Cys Leu Gln Tyr Pro Phe Thr Ser Val Pro Leu Ser
 370 375 380
 Gly Gly Ala Pro Gly Thr Arg Ala Ser Arg Arg Cys Asp Arg Ala Gly
 385 390 395 400
 Arg Trp Glu Pro Gly Asp Tyr Ser His Cys Leu Tyr Thr Asn Asp Ile
 405 410 415
 Thr Arg Val Leu Tyr Thr Phe Val Leu Met Pro Ile Asn Ala Ser Asn
 420 425 430
 Ala Leu Thr Leu Ala His Gln Leu Arg Val Tyr Thr Ala Glu Ala Ala
 435 440 445
 Ser Phe Ser Asp Met Met Asp Val Val Tyr Val Ala Gln Met Ile Gln
 450 455 460
 Lys Phe Leu Gly Tyr Val Asp Gln Ile Lys Glu Leu Val Glu Val Met
 465 470 475 480
 Val Asp Met Ala Ser Asn Leu Met Leu Val Asp Glu His Leu Leu Trp
 485 490 495
 Leu Ala Gln Arg Glu Asp Lys Ala Cys Ser Gly Ile Val Gly Ala Leu
 500 505 510
 Glu Arg Ile Gly Gly Ala Ala Leu Ser Pro His Ala Gln His Ile Ser
 515 520 525
 Val Asn Ser Arg Asn Val Ala Leu Glu Ala Tyr Leu Ile Lys Pro His
 530 535 540
 Ser Tyr Val Gly Leu Thr Cys Thr Ala Phe Gln Arg Arg Glu Val Gly
 545 550 555 560
 Val Ser Gly Ala Gln Pro Ser Ser Val Gly Gln Asp Ala Pro Val Glu
 565 570 575
 Pro Glu Pro Leu Ala Asp Gln Gln Leu Arg Phe Arg Cys Thr Thr Gly
 580 585 590
 Arg Pro Asn Ile Ser Leu Ser Ser Phe His Ile Lys Asn Ser Val Ala
 595 600 605
 Leu Ala Ser Ile Gln Leu Pro Pro Ser Leu Phe Ser Thr Leu Pro Ala
 610 615 620
 Ala Leu Ala Pro Pro Val Pro Pro Asp Cys Thr Leu Gln Leu Leu Val
 625 630 635 640
 Phe Arg Asn Gly Arg Leu Phe Arg Ser His Gly Asn Asn Thr Ser Arg
 645 650 655
 Pro Gly Ala Ala Gly Pro Gly Lys Arg Arg Gly Val Ala Thr Pro Val
 660 665 670
 Ile Phe Ala Gly Thr Ser Gly Cys Gly Val Gly Asn Leu Thr Glu Pro
 675 680 685
 Val Ala Val Ser Leu Arg His Trp Ala Glu Gly Ala Asp Pro Met Ala
 690 695 700
 Ala Trp Trp Asn Gln Asp Gly Pro Gly Gly Trp Ser Ser Glu Gly Cys
 705 710 715 720
 Arg Leu Arg Tyr Ser Gln Pro Asn Val Ser Ser Leu Tyr Cys Gln His
 725 730 735
 Leu Gly Asn Val Ala Val Leu Met Glu Leu Asn Ala Phe Pro Arg Glu
 740 745 750
 Ala Gly Gly Ser Gly Ala Gly Leu His Pro Val Val Tyr Pro Cys Thr
 755 760 765
 Ala Leu Leu Leu Cys Leu Phe Ser Thr Ile Ile Thr Tyr Ile Leu
 770 775 780
 Asn His Ser Ser Ile His Val Ser Arg Lys Gly Trp His Met Leu Leu
 785 790 795 800
 Asn Leu Cys Phe His Met Ala Met Thr Ser Ala Val Phe Val Gly Gly
 805 810 815
 Val Thr Leu Thr Asn Tyr Gln Met Val Cys Gln Ala Val Gly Ile Thr
 820 825 830
 Leu His Tyr Ser Ser Leu Ser Ser Leu Leu Trp Met Gly Val Lys Ala
 835 840 845
 Arg Val Leu His Lys Glu Leu Ser Trp Arg Ala Pro Pro Leu Glu Glu
 850 855 860
 Gly Glu Ala Ala Pro Pro Gly Pro Arg Pro Met Leu Arg Phe Tyr Leu
 865 870 875 880
 Ile Ala Gly Gly Ile Pro Leu Ile Ile Cys Gly Ile Thr Ala Ala Val
 885 890 895

Asn Ile His Asn Tyr Arg Asp His Ser Pro Tyr Cys Trp Leu Val Trp
 900 905 910
 Arg Pro Ser Leu Gly Ala Phe Tyr Ile Pro Val Ala Leu Ile Leu Pro
 915 920 925
 Ile Thr Trp Ile Tyr Phe Leu Cys Ala Gly Leu His Leu Arg Ser His
 930 935 940
 Val Ala Gln Asn Pro Lys Gln Gly Asn Arg Ile Ser Leu Glu Pro Gly
 945 950 955 960
 Glu Glu Leu Arg Gly Ser Thr Arg Leu Arg Ser Ser Gly Val Leu Leu
 965 970 975
 Asn Asp Ser Gly Ser Leu Leu Ala Thr Val Ser Ala Gly Val Gly Thr
 980 985 990
 Pro Ala Pro Pro Glu Asp Gly Asp Gly Val Tyr Ser Pro Gly Val Gln
 995 1000 1005
 Leu Gly Ala Leu Met Thr Thr His Phe Leu Tyr Leu Ala Met Trp Ala
 1010 1015 1020
 Cys Gly Ala Leu Ala Val Ser Gln Arg Trp Leu Pro Arg Val Val Cys
 1025 1030 1035 1040
 Ser Cys Leu Tyr Gly Val Ala Ala Ser Ala Leu Gly Leu Phe Val Phe
 1045 1050 1055
 Thr His His Cys Ala Arg Arg Arg Asp Val Arg Ala Ser Trp Arg Ala
 1060 1065 1070
 Cys Cys Pro Pro Ala Ser Pro Ser Ala Ser His Val Pro Ala Arg Ala
 1075 1080 1085
 Leu Pro Thr Ala Thr Glu Asp Gly Ser Pro Val Leu Gly Glu Gly Pro
 1090 1095 1100
 Ala Ser Leu Lys Ser Ser Pro Ser Gly Ser Ser Gly Arg Ala Pro Pro
 1105 1110 1115 1120
 Pro Pro Cys Lys Leu Thr Asn Leu Gln Val Ala Gln Ser Gln Val Cys
 1125 1130 1135
 Glu Ala Ser Val Ala Ala Arg Gly Asp Gly Glu Pro Glu Pro Thr Gly
 1140 1145 1150
 Ser Arg Gly Ser Leu Ala Pro Arg His His Asn Asn Leu His His Gly
 1155 1160 1165
 Arg Arg Val His Lys Ser Arg Ala Lys Gly His Arg Ala Gly Glu Thr
 1170 1175 1180
 Gly Gly Lys Ser Arg Leu Lys Ala Leu Arg Ala Gly Thr Ser Pro Gly
 1185 1190 1195 1200
 Ala Pro Glu Leu Leu Ser Ser Glu Ser Gly Ser Leu His Asn Ser Pro
 1205 1210 1215
 Ser Asp Ser Tyr Pro Gly Ser Ser Arg Asn Ser Pro Gly Asp Gly Leu
 1220 1225 1230
 Pro Leu Glu Gly Glu Pro Met Leu Thr Pro Ser Glu Gly Ser Asp Thr
 1235 1240 1245
 Ser Ala Ala Pro Ile Ala Glu Thr Gly Arg Pro Gly Gln Arg Arg Ser
 1250 1255 1260
 Ala Ser Arg Asp Asn Leu Lys Gly Ser Gly Ser Ala Leu Glu Arg Glu
 1265 1270 1275 1280
 Ser Lys Arg Arg Ser Tyr Pro Leu Asn Thr Thr Ser Leu Asn Gly Ala
 1285 1290 1295
 Pro Lys Gly Gly Lys Tyr Glu Asp Ala Ser Val Thr Gly Ala Glu Ala
 1300 1305 1310
 Ile Ala Gly Gly Ser Met Lys Thr Gly Leu Trp Lys Ser Glu Thr Thr
 1315 1320 1325
 Val

<210> 192

<211> 500

<212> PRT

<213> Mus musculus

<400> 192

Met Arg Ala Gln Leu Trp Leu Leu Gln Leu Leu Leu Arg Gly Ala
 1 5 10 15
 Ala Arg Ala Leu Ser Pro Ala Thr Pro Ala Gly His Asn Glu Gly Gln

	20		25		30
Asp	Ser	Ala	Trp	Thr	Ala
	35				
Pro	Arg	Glu	Ser	Pro	Ala
	50				
Ser	Gln	Asp	Leu	Gly	Gly
	65				
Asn	Arg	Thr	Arg	Val	Val
	85				
Val	Tyr	Gly	Pro	Gly	Glu
	100				
Ala	Val	Ala	Asn	Arg	Ser
	115				
Ser	His	Arg	Gln	Ala	Ser
	130				
Tyr	Gly	His	Pro	Leu	Arg
	145				
Phe	Met	Gly	Asp	Met	Leu
	165				
Ala	Pro	Leu	Met	Ala	Asn
	180				
Val	Ala	Tyr	Phe	Asp	Asn
	195				
Val	Tyr	Leu	Gln	Asp	Arg
	210				
Ala	Leu	His	Arg	Asp	Gly
	225				
Met	Ala	Val	Leu	Asp	Ile
	245				
Leu	Ser	Asp	Ala	Phe	Met
	260				
Ser	Gln	Arg	Thr	Ile	Phe
	275				
Ser	Lys	Ile	Thr	Thr	Thr
	290				
Cys	Leu	Gln	His	Gln	Ser
	305				
Phe	Asn	Cys	Ser	Trp	Cys
	325				
Asp	Arg	Tyr	Arg	Gln	Glu
	340				
Glu	Gly	Lys	Thr	Cys	Glu
	355				
Ser	Pro	Asp	Ser	Ser	Phe
	370				
Ser	Ser	Leu	Phe	Ile	Asp
	385				
Asn	Pro	Tyr	Ala	Glu	Gly
	405				
Ser	Lys	Gly	Pro	Pro	Val
	420				
Ala	Val	Leu	Leu	Val	Ala
	435				
Gly	His	Pro	Asn	Ser	Asn
	450				
His	His	Trp	Pro	Ala	Met
	465				
Tyr	Thr	Glu	Val	Glu	Pro
	485				
Ala	Glu	Gln	Cys		
	500				

<210> 193

<211> 530

<212> PRT

<213> Mus musculus

<400> 193

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Met Ala Arg Phe Arg Arg Ala Asp Leu Ala Ala Ala Gly Val Met Leu
1      5      10      15
Leu Cys His Phe Leu Thr Asp Arg Phe His Phe Ala His Gly Glu Pro
20     25     30
Gly His His Thr Asn Asp Trp Ile Tyr Glu Val Thr Asn Ala Phe Pro
35     40     45
Trp Asn Glu Glu Gly Val Glu Val Asp Ser Gln Ala Tyr Asn His Arg
50     55     60
Trp Lys Arg Asn Val Asp Pro Phe Lys Ala Val Asp Thr Asn Arg Ala
65     70     75     80
Ser Met Gly Gln Ala Ser Pro Glu Ser Lys Gly Phe Thr Asp Leu Leu
85     90     95
Leu Asp Asp Gly Gln Asp Asn Asn Thr Gln Ile Glu Glu Asp Thr Asp
100    105    110
His Asn Tyr Tyr Ile Ser Arg Ile Tyr Gly Pro Ala Asp Ser Ala Ser
115    120    125
Arg Asp Leu Trp Val Asn Ile Asp Gln Met Glu Lys Asp Lys Val Lys
130    135    140
Ile His Gly Ile Leu Ser Asn Thr His Arg Gln Ala Ala Arg Val Asn
145    150    155    160
Leu Ser Phe Asp Phe Pro Phe Tyr Gly His Phe Leu Asn Glu Val Thr
165    170    175
Val Ala Thr Gly Gly Phe Ile Tyr Thr Gly Glu Val Val His Arg Met
180    185    190
Leu Thr Ala Thr Gln Tyr Ile Ala Pro Leu Met Ala Asn Phe Asp Pro
195    200    205
Ser Val Ser Arg Asn Ser Thr Val Arg Tyr Phe Asp Asn Gly Thr Ala
210    215    220
Leu Val Val Gln Trp Asp His Val His Leu Gln Asp Asn Tyr Asn Leu
225    230    235    240
Gly Ser Phe Thr Phe Gln Ala Thr Leu Leu Met Asp Gly Arg Ile Ile
245    250    255
Phe Gly Tyr Lys Glu Ile Pro Val Leu Val Thr Gln Ile Ser Ser Thr
260    265    270
Asn His Pro Val Lys Val Gly Leu Ser Asp Ala Phe Val Val Val His
275    280    285
Arg Ile Gln Gln Ile Pro Asn Val Arg Arg Arg Thr Ile Tyr Glu Tyr
290    295    300
His Arg Val Glu Leu Gln Met Ser Lys Ile Thr Asn Ile Ser Ala Val
305    310    315    320
Glu Met Thr Pro Leu Pro Thr Cys Leu Gln Phe Asn Gly Cys Gly Pro
325    330    335
Cys Val Ser Ser Gln Ile Gly Phe Asn Cys Ser Trp Cys Ser Lys Leu
340    345    350
Gln Arg Cys Ser Ser Gly Phe Asp Arg His Arg Gln Asp Trp Val Asp
355    360    365
Ser Gly Cys Pro Glu Glu Val Gln Ser Lys Glu Lys Met Cys Glu Lys
370    375    380
Thr Glu Pro Gly Glu Thr Ser Gln Thr Thr Thr Thr Ser His Thr Thr
385    390    395    400
Thr Met Gln Phe Arg Val Leu Thr Thr Thr Arg Arg Ala Val Thr Ser
405    410    415
Gln Met Pro Thr Ser Leu Pro Thr Glu Asp Asp Thr Lys Ile Ala Leu
420    425    430
His Leu Lys Asp Ser Gly Ala Ser Thr Asp Asp Ser Ala Ala Glu Lys
435    440    445
Lys Gly Gly Thr Leu His Ala Gly Leu Ile Val Gly Ile Leu Ile Leu
450    455    460
Val Leu Ile Ile Ala Ala Ala Ile Leu Val Thr Val Tyr Met Tyr His
465    470    475    480
His Pro Thr Ser Ala Ala Ser Ile Phe Phe Ile Glu Arg Arg Pro Ser
485    490    495
Arg Trp Pro Ala Met Lys Phe Arg Arg Gly Ser Gly His Pro Ala Tyr
500    505    510

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Ala Glu Val Glu Pro Val Gly Glu Lys Glu Gly Phe Ile Val Ser Glu
 515 520 525
 Gln Cys
 530

<210> 194
 <211> 562
 <212> PRT
 <213> Mus musculus

<400> 194
 Met Asp Arg Ala Gly Arg Leu Gly Ala Gly Leu Arg Gly Leu Cys Val
 1 5 10 15
 Ala Ala Leu Val Leu Val Cys Ala Gly His Gly Gly Arg Arg Glu Asp
 20 25 30
 Gly Gly Pro Ala Cys Tyr Gly Gly Phe Asp Leu Tyr Phe Ile Leu Asp
 35 40 45
 Lys Ser Gly Ser Val Leu His His Trp Asn Glu Ile Tyr Tyr Phe Val
 50 55 60
 Glu Gln Leu Ala His Arg Phe Ile Ser Pro Gln Leu Arg Met Ser Phe
 65 70 75 80
 Ile Val Phe Ser Thr Arg Gly Thr Thr Leu Met Lys Leu Thr Glu Asp
 85 90 95
 Arg Glu Gln Ile Arg Gln Gly Leu Glu Glu Leu Gln Lys Val Leu Pro
 100 105 110
 Gly Gly Asp Thr Tyr Met His Glu Gly Phe Glu Arg Ala Ser Glu Gln
 115 120 125
 Ile Tyr Tyr Glu Asn Ser Gln Gly Tyr Arg Thr Ala Ser Val Ile Ile
 130 135 140
 Ala Leu Thr Asp Gly Glu Leu His Glu Asp Leu Phe Phe Tyr Ser Glu
 145 150 155 160
 Arg Glu Ala Asn Arg Ser Arg Asp Leu Gly Ala Ile Val Tyr Cys Val
 165 170 175
 Gly Val Lys Asp Phe Asn Glu Thr Gln Leu Ala Arg Ile Ala Asp Ser
 180 185 190
 Lys Asp His Val Phe Pro Val Asn Asp Gly Phe Gln Ala Leu Gln Gly
 195 200 205
 Ile Ile His Ser Ile Leu Lys Lys Ser Cys Ile Glu Ile Leu Ala Ala
 210 215 220
 Glu Pro Ser Thr Ile Cys Ala Gly Glu Ser Phe Gln Val Val Val Arg
 225 230 235 240
 Gly Asn Gly Phe Arg His Ala Arg Asn Val Asp Arg Val Leu Cys Ser
 245 250 255
 Phe Lys Ile Asn Asp Ser Val Thr Leu Asn Glu Lys Pro Phe Ala Val
 260 265 270
 Glu Asp Thr Tyr Leu Leu Cys Pro Ala Pro Ile Leu Lys Glu Val Gly
 275 280 285
 Met Lys Ala Ala Leu Gln Val Ser Met Asn Asp Gly Leu Ser Phe Ile
 290 295 300
 Ser Ser Ser Val Ile Ile Thr Thr Thr His Cys Ser Asp Gly Ser Ile
 305 310 315 320
 Leu Ala Ile Ala Leu Leu Val Leu Phe Leu Leu Ala Leu Ala Leu
 325 330 335
 Leu Trp Trp Phe Trp Pro Leu Cys Cys Thr Val Ile Ile Lys Glu Val
 340 345 350
 Pro Pro Pro Pro Val Glu Glu Ser Glu Glu Glu Asp Asp Gly Leu
 355 360 365
 Pro Lys Lys Lys Trp Pro Thr Val Asp Ala Ser Tyr Tyr Gly Gly Arg
 370 375 380
 Gly Val Gly Gly Ile Lys Arg Met Glu Val Arg Trp Gly Glu Lys Gly
 385 390 395 400
 Ser Thr Glu Glu Gly Ala Lys Leu Glu Lys Ala Lys Asn Ala Arg Val
 405 410 415
 Lys Met Pro Glu Gln Glu Tyr Glu Phe Pro Glu Pro Arg Asn Leu Asn
 420 425 430
 Asn Asn Met Arg Arg Pro Ser Ser Pro Arg Lys Trp Tyr Ser Pro Ile

	435		440		445		
Lys	Gly	Lys	Leu	Asp	Ala	Leu	Trp
450							
Arg	Val	Ser	Val	Met	Arg	Pro	Gln
465							
Asn	Phe	Thr	Arg	Val	Lys	Asn	Ser
Asn	Thr	Tyr	His	Pro	Ser	Ser	Pro
Pro	Pro	Pro	Ala	Pro	His	Cys	Pro
Pro	Pro	Ile	Pro	Ser	Pro	Pro	Thr
Pro	Pro	Pro	Asn	Arg	Ala	Pro	Pro
Ser	Val						

<210> 195

<211> 2565

<212> DNA

<213> Homo sapiens

<400> 195

tcgcgatgct gctgcgcctg ttgctggcct gggcgggcgc agggccca ctgggccagg
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 acccctgggc tgctgagccc cgtgcgcctt gggcccccag cagctgtac gctctcttc
 120
 cacggcgccg caccttctg gaggcctggc gggcctggcg cagctgggg ggcgacctgg
 180
 caactctcgg gaccccgag gaggccagc gtgtggacag cctggtgggt gggggccca
 240
 ccagccggct gctgtggatc gggctgcagc ggcaggcccg gcaatggcag ctgcagcgcc
 300
 caactgcgcg cttcacgtgg accacagggg accaggacac ggccttcacc aactggggcc
 360
 agccagcctc tggaggcccc tgccggcccc agcgtgtgtt ggcctggag gcaagtggcg
 420
 agcacccgtg gctggagggg tcgtgcacgc tggctgtcga cggctacctg tggcagttt
 480
 gcttcgaggg cgcctgcccg gcgtgcaag atgaggccgg ccaggccggc ccagccgtgt
 540
 ataccacgcc cttccacctg gtctccacag agtttgagtg gctgcccttc ggtctgtgtg
 600
 ccgtgtgca gtgccaggct ggcagggggg cctctctgct ctgcgtgaag cagcctgagg
 660
 gaggtgtggg ctggtcacgg gctggggccc tgtgctggg gactggctgc agccctgaca
 720
 acggggggctg cgaacacgaa tgtgtggagg aggtggatgg tcacgtgtcc tgccgtgca
 780
 ctgagggcct ccggctggca gcagacgggc gcaagttcga ggaccctgt gccacggctc
 840
 cgtgcagaca gcagtggtgag ccgggtgggc cacaaggcta cagctgccac tgtgcctgg
 900
 gtttccggcc agcggaggat gatccgcacc gctgtgtgga cacagatgag tgccagattg
 960
 ccggtgtgtg ccagcagatg tgtgtcaact acgttggtgg cttcagatgt tattgtagcg
 1020
 agggacatga gctggaggct gatggcatca gctgcagccc tgcagggggc atgggtgcc
 1080
 aggcctccca ggacctcgga gatgagttgc tggatgacgg ggaggatgag gaagatgaag
 1140
 acgagggcct gaaggccttc aacgggtggc ggacggagat gcctggggatc ctgtggatgg
 1200
 agcctacgca gccgcctgac ttgcccctgg cctatagacc gagcttccca gaggacagag
 1260

agccacagat accctacccg gagccacact gggccacccc gctcagtgcc ccaggggtcc
 1320
 cctaccactc ctcagtgctc tccgtcaccc ggccgtgtggt ggtctctgcc acgcatccca
 1380
 cactgccttc tgcccaccag cctcctgtga tccctgccac acaccagct ttgtcccggt
 1440
 accaccagat ccccgatgc gcagccaact atccagatct gccttctgcc taccacccg
 1500
 gtattctctc tgtctctcat tcagcacagc ctcccgccca ccagccccct atgatctcaa
 1560
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<211> 757

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<213> Homo sapiens

<400> 196

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<211> 266

<212> PRT

<213> Homo sapiens

<400> 198

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 Ser Val Gly Gln Asp Ser Pro Glu Pro Arg Ser Phe Thr Asp Leu Leu
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 His Asn Tyr Tyr Ile Ser Arg Ile Tyr Gly Pro Ser Asp Ser Ala Ser

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 265 270 275
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<211> 350

<212> PRT

<213> Homo sapiens

<400> 202

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<211> 7540
<212> DNA
<213> Homo sapiens

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 Asn Thr Phe Gln Ala Val Leu Ala Ser Ser Asp Ser Ser Tyr Ala
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<213> Homo sapiens

<400> 226

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<211> 1202

<212> PRT

<213> Homo sapiens

<400> 228

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 35 40 45
 Gly Lys Val Glu Leu Val Phe Ser Ala Thr Pro Glu Lys Ile Gln Gly
 50 55 60
 Ser Glu His Leu Tyr Asn Asp His Gly Val Ile Val Asp Tyr Asn Thr
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 Thr Asp Pro Leu Ile Arg Trp Asp Ser Tyr Glu Asn Leu Ser Ala Asp
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 Gly Glu Val Leu His Thr Gln Gly Pro Val Asp Gly Ser Leu Tyr Ala
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 Lys Val Arg Lys Lys Ser Ser Ser Asp Pro Gly Ile Pro Gly Gly Pro
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 Gln Ala Ile Pro Ala Thr Asn Ser Pro Asp His Ser Asp His Thr Leu
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 Lys Thr Glu Glu Arg Leu Ala Pro Gly Thr Arg Arg Gly Leu Ser Ala
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 Asp Pro Gly Ser Ser Leu Lys Glu Met Thr Asp Ala Arg Ser Lys Tyr
 195 200 205
 Ser Gly Thr Arg His Val Val Pro Ala Gln Val His Val Asn Gly Asp
 210 215 220
 Ala Ala Leu Lys Asp Arg Glu Thr Asp Ile Leu Asp Asp Glu Met Pro
 225 230 235 240
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 245 250 255
 Glu Gly Pro Gln Ser Ala His Leu Gly Pro Phe Thr Cys His Lys Ser
 260 265 270
 Ser Gln Asn Ser Leu Leu Ser Asp Gly Phe Gly Ser Asn Val Gly Glu
 275 280 285
 Asp Pro Gln Gly Thr Leu Val Pro Asp Leu Gly Leu Met Asp Gly
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 Pro Tyr Glu Arg Glu Arg Thr Phe Gly Ser Arg Glu Pro Lys Gln Pro
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 Gln Pro Leu Leu Arg Lys Pro Ser Val Ser Ala Gln Met Gln Ala Tyr
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 Val Ser Arg Cys Pro Ala Asp Asn Pro Gly Leu Val Gln Ala Gln Pro

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Ser Pro Ser Lys Ala Phe Lys Pro Arg Phe Pro Gly Asp Gln Val Val			
	420	425	430
Asn Gly Ala Gly Pro Glu Leu Ser Thr Gly Pro Ser Pro Gly Ser Pro			
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Thr Leu Asp Ile Asp Gln Ser Ile Glu Glu Leu Asn Arg Leu Ile Leu			
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Glu Leu Asp Pro Thr Phe Glu Pro Ile Pro Thr His Met Asn Ala Leu			
465	470	475	480
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Leu Arg Ala Ser Ser Arg Leu Pro Asp Thr Gly Glu Gly Pro Ser Arg			
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Glu Pro Arg Ser Cys Pro Glu Thr Leu Thr His Ala Val Gly Met Ser			
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Glu Ser Pro Ile Gly Pro Lys Ser Thr Met Leu Arg Ala Asp Ala Ser			
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Ser Thr Pro Ser Phe Gln Gln Ala Phe Ala Ser Ser Cys Thr Ile Ser			
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Ser Asn Gly Pro Gly Gln Arg Arg Glu Ser Ser Ser Ser Ala Glu Arg			
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Gln Trp Val Glu Ser Ser Pro Lys Pro Met Val Ser Leu Leu Gly Ser			
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Gly Arg Pro Thr Gly Ser Pro Leu Ser Ala Glu Phe Ser Gly Thr Arg			
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Lys Asp Ser Pro Val Leu Ser Cys Phe Pro Pro Ser Glu Leu Gln Ala			
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Pro Phe His Ser His Glu Leu Ser Leu Ala Glu Pro Pro Asp Ser Leu			
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Ala Pro Pro Ser Ser Gln Ala Phe Leu Gly Phe Gly Thr Ala Pro Val			
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Ala Ala Asp Asn Gly Phe Leu Ser His Asn Phe Leu Thr Val Ala Pro			
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Gly His Ser Ser His His Ser Pro Gly Leu Gln Gly Gln Gly Val Thr			
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Leu Pro Gly Gln Pro Pro Leu Pro Glu Lys Lys Arg Ala Ser Glu Gly			
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Asp Arg Ser Leu Gly Ser Val Ser Pro Ser Ser Ser Gly Phe Ser Ser			
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Pro His Ser Gly Ser Thr Ile Ser Ile Pro Phe Pro Asn Val Leu Pro			
	885	890	895
Asp Phe Ser Lys Ala Ser Glu Ala Ala Ser Pro Leu Pro Asp Ser Pro			

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 930 935 940
 Asp Lys Glu Pro Gly Ser Phe Ile Val Arg Asp Ser His Ser Phe Arg
 945 950 955 960
 Gly Ala Tyr Gly Leu Ala Met Lys Val Ala Thr Pro Pro Pro Ser Val
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 1090 1095 1100
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<211> 2320

<212> DNA

<213> Homo sapiens

<400> 229

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<212> FRT

<213> Homo sapiens

<400> 230

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Arg Glu Ser Pro Gly His Val Ser Glu Pro Asp Arg Thr Gln Leu Ser			
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Gln Asp Leu Gly Gly Gly Thr Leu Ala Met Asp Thr Leu Pro Asp Asn			
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Arg Thr Arg Val Val Glu Asp Asn His Ser Tyr Tyr Val Ser Arg Leu			
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Tyr Gly Pro Ser Glu Pro His Ser Arg Glu Leu Trp Val Asp Val Ala			
100	105	110	
Glu Ala Asn Arg Ser Gln Val Lys Ile His Thr Ile Leu Ser Asn Thr			
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His Arg Gln Ala Ser Arg Val Val Leu Ser Phe Asp Phe Pro Phe Tyr			
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Gly His Pro Leu Arg Gln Ile Thr Ile Ala Thr Gly Gly Phe Ile Phe			
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Met Gly Asp Val Ile His Arg Met Leu Thr Ala Thr Gln Tyr Val Ala			
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Pro Leu Met Ala Asn Phe Asn Pro Gly Tyr Ser Asp Asn Ser Thr Val			
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Val Tyr Phe Asp Asn Gly Thr Val Phe Val Val Gln Trp Asp His Val			
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Tyr Leu Gln Gly Trp Glu Asp Lys Gly Ser Phe Thr Phe Gln Ala Ala			
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Leu His His Asp Gly Arg Ile Val Phe Ala Tyr Lys Glu Ile Pro Met			
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Lys Val Thr Ser Met Ser Ala Val Glu Phe Thr Pro Leu Pro Thr Cys			
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Leu Gln His Arg Ser Cys Asp Ala Cys Met Ser Ser Asp Leu Thr Phe			
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Arg Tyr Arg Gln Glu Trp Met Asp Tyr Gly Cys Ala Gln Glu Ala Glu			
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Gly Arg Met Cys Glu Asp Phe Gln Asp Glu Asp His Asp Ser Ala Ser			
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Pro Asp Thr Ser Phe Ser Pro Tyr Asp Gly Asp Leu Thr Thr Ser			
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Ser Ser Leu Phe Ile Asp Ser Leu Thr Thr Glu Asp Asp Thr Lys Leu			
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Gly His Pro Thr Ser Asn Ala Ala Leu Phe Phe Ile Glu Arg Arg Pro			
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His His Trp Pro Ala Met Lys Phe Arg Ser His Pro Asp His Ser Thr			
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<212> DNA
 <213> Homo sapiens

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 225 230 235 240
 Leu Cys Met Ala Pro Val His Leu Arg Gly Phe Asn Val Ala Asp Val
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 Cys Asn Ala Asn Ser Ile Ser Cys Pro Ser Pro Cys Thr Cys Ser Asn
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<212> DNA

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 Cys Arg Asp Gln Leu Ser Ala Asp Met Tyr Ser Phe Val Ala Lys Glu
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 Pro Leu Met Thr Phe Glu Leu Tyr Asp Glu Trp Ile Gln Ala Ser Asn
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 <213> Homo sapiens

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 Gly Ala Ser Gly Tyr Pro Gly Asn Pro Gly Leu Pro Gly Ile Pro Gly
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<212> DNA
<213> Homo sapiens
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Pro Thr Met Tyr Thr Phe Arg Pro Trp Thr Ile Arg Gln Tyr Ala Gly
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145          150          155          160
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Lys Leu Arg Ile Glu Glu Cys Ala Ala Arg Arg Gln Ala Arg Ile Asp

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Phe	Lys	Ile	Ile	Asp	Glu	Asn	Thr	Val	His	Met	Ser	Trp	Ala	Glu	Pro
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Gly	Pro	Thr	Lys	Glu	Phe	Thr	Leu	Ser	Ala	Ser	Thr	Thr	Glu	Thr	Leu
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Leu	Ser	Glu	Leu	Val	Pro	Glu	Thr	Glu	Tyr	Val	Val	Thr	Ile	Thr	Ser
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Tyr	Asp	Glu	Val	Glu	Glu	Ser	Val	Pro	Val	Ile	Gly	Gln	Leu	Thr	Ile
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Gln	Thr	Gly	Ser	Ser	Thr	Lys	Pro	Val	Glu	Lys	Lys	Pro	Gly	Lys	Thr
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Asp Thr Lys Tyr Glu Val Ser Val Ile Pro Glu Tyr Phe Ser Gly Pro		780
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 Asp Gly Gly Arg Thr Ser Asp Thr Gly Arg Thr Leu Met Arg Gly Leu
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 Ala Arg Asn Val Gln Val Tyr Asn Pro Thr Pro Asn Arg Leu Gly Val
 1940 1945 1950
 Arg Trp Asp Pro Ala Pro Gly Pro Val Leu Gln Tyr Arg Val Val Tyr
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 Ser Pro Val Asp Gly Thr Arg Pro Ser Glu Ser Ile Val Val Pro Gly
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 Asn Thr Arg Met Val His Leu Glu Arg Leu Ile Pro Asp Thr Leu Tyr
 1985 1990 1995 2000
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 2065 2070 2075 2080
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 Thr Val Gly Leu Leu Pro Pro Gln Asn Ile His Ile Ser Asp Glu Trp

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Ser Val Pro Leu	Thr Asp Gln Gly Thr	Leu Tyr Leu Asn Val Thr
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Asp Leu Lys Thr	Tyr Gln Ile Gly Trp	Asp Thr Phe Cys Val Lys Trp
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Ser Pro His Arg	Ala Ala Thr Ser	Tyr Arg Leu Lys Leu Ser Pro Ala
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Asp Gly Thr Arg	Gly Gln Glu Ile	Thr Val Arg Gly Ser Glu Thr Ser
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His Cys Phe Thr	Gly Leu Ser Pro	Asp Thr Asp Tyr Gly Val Thr Val
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His Thr Thr Val	Lys Pro Thr Glu Ala	Pro Thr Thr Pro
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Pro Pro Pro Pro	Thr Ile Pro Pro	Ala Arg Asp Val Cys Lys Gly Ala
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Lys Ala Asp Ile	Val Phe Leu Thr	Asp Ala Ser Trp Ser Ile Gly Asp
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Asp Asn Phe Asn	Lys Val Val Lys	Phe Ile Phe Asn Thr Val Gly Gly
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Phe Asp Glu Ile	Ser Pro Ala Gly	Ile Gln Val Ser Phe Val Gln Tyr
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Gln Gln Ser Gly	Phe Ser Val Phe	Val Val Ala Asp Val Asp
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<213> Homo sapiens

<400> 259

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 Glu Ala Ser Gln Asn Ile Thr Tyr His Cys Lys Asn Ser Val Ala Tyr
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 Met Asp Gln Gln Thr Gly Asn Leu Lys Lys Ala Leu Leu Lys Gly
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 Ser Asn Glu Ile Glu Ile Arg Ala Glu Gly Asn Ser Arg Phe Thr Tyr
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 Ser Val Thr Val Asp Gly Cys Thr Ser His Thr Gly Ala Trp Gly Lys
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 Thr Val Ile Glu Tyr Lys Thr Thr Lys Ser Ser Arg Leu Pro Ile Ile
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<212> DNA
<213> Homo sapiens

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<211> 412

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<400> 263

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 Glu Glu Met His Gly Glu Arg Glu Glu Gly Cys Thr Gln Glu Asn Thr
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 Glu Ser Glu Tyr Tyr Ala Lys Glu Ile His Lys Phe Asp Met Ile Gln
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 115 120 125
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 Ser Lys Arg Asn Glu Gln Arg Ile Glu Leu Phe Gln Ile Leu Arg Pro
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400> 264
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<211> 1366

<212> PRT

<213> Homo sapiens

<400> 265

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 Thr Pro Gly Gln Thr Gly Ala Arg Gly Leu Pro Gly Glu Arg Gly Arg
 210 215 220
 Val Gly Ala Pro Gly Pro Ala Gly Ala Arg Gly Ser Asp Gly Ser Val
 225 230 235 240
 Gly Pro Val Gly Pro Ala Gly Pro Ile Gly Ser Ala Gly Pro Pro Gly
 245 250 255
 Phe Pro Gly Ala Pro Gly Pro Lys Gly Glu Ile Gly Ala Val Gly Asn
 260 265 270
 Ala Gly Pro Ala Gly Pro Ala Gly Pro Arg Gly Glu Val Gly Leu Pro
 275 280 285
 Gly Leu Ser Gly Pro Val Gly Pro Pro Gly Asn Pro Gly Ala Asn Gly
 290 295 300
 Leu Thr Gly Ala Lys Gly Ala Ala Gly Leu Pro Gly Val Ala Gly Ala
 305 310 315 320
 Pro Gly Leu Pro Gly Pro Arg Gly Ile Pro Gly Pro Val Gly Ala Ala
 325 330 335
 Gly Ala Thr Gly Ala Arg Gly Leu Val Gly Glu Pro Gly Pro Ala Gly
 340 345 350
 Ser Lys Gly Glu Ser Gly Asn Lys Gly Glu Pro Gly Ser Ala Gly Pro
 355 360 365
 Gln Gly Pro Pro Gly Pro Ser Gly Glu Glu Gly Lys Arg Gly Pro Asn
 370 375 380
 Gly Glu Ala Gly Ser Ala Gly Pro Pro Gly Pro Pro Gly Leu Arg Gly
 385 390 395 400
 Ser Pro Gly Ser Arg Gly Leu Pro Gly Ala Asp Gly Arg Ala Gly Val

405 410 415
 Met Gly Pro Pro Gly Ser Arg Gly Ala Ser Gly Pro Ala Gly Val Arg
 420 425 430
 Gly Pro Asn Gly Asp Ala Gly Arg Pro Gly Glu Pro Gly Leu Met Gly
 435 440 445
 Pro Arg Gly Leu Pro Gly Ser Pro Gly Asn Ile Gly Pro Ala Gly Lys
 450 455 460
 Glu Gly Pro Val Gly Leu Pro Gly Ile Asp Gly Arg Pro Gly Pro Ile
 465 470 475 480
 Gly Pro Ala Gly Ala Arg Gly Glu Pro Gly Asn Ile Gly Phe Pro Gly
 485 490 495
 Pro Lys Gly Pro Thr Gly Asp Pro Gly Lys Asn Gly Asp Lys Gly His
 500 505 510
 Ala Gly Leu Ala Gly Ala Arg Gly Ala Pro Gly Pro Asp Gly Asn Asn
 515 520 525
 Gly Ala Gln Gly Pro Pro Gly Pro Gln Gly Val Gln Gly Gly Lys Gly
 530 535 540
 Glu Gln Gly Pro Ala Gly Pro Pro Gly Phe Gln Gly Leu Pro Gly Pro
 545 550 555 560
 Ser Gly Pro Ala Gly Glu Val Gly Lys Pro Gly Glu Arg Gly Leu His
 565 570 575
 Gly Glu Phe Gly Leu Pro Gly Pro Ala Gly Pro Arg Gly Glu Arg Gly
 580 585 590
 Pro Pro Gly Glu Ser Gly Ala Ala Gly Pro Thr Gly Pro Ile Gly Ser
 595 600 605
 Arg Gly Pro Ser Gly Pro Pro Gly Pro Asp Gly Asn Lys Gly Glu Pro
 610 615 620
 Gly Val Val Gly Ala Val Gly Thr Ala Gly Pro Ser Gly Pro Ser Gly
 625 630 635 640
 Leu Pro Gly Glu Arg Gly Ala Ala Gly Ile Pro Gly Gly Lys Gly Glu
 645 650 655
 Lys Gly Glu Pro Gly Leu Arg Gly Glu Ile Gly Asn Pro Gly Arg Asp
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 675 680 685
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 690 695 700
 Ala Gly Pro Arg Gly Ser Pro Gly Glu Arg Gly Glu Val Gly Pro Ala
 705 710 715 720
 Gly Pro Asn Gly Phe Ala Gly Pro Ala Gly Ala Gly Gln Pro Gly
 725 730 735
 Ala Lys Gly Glu Arg Gly Ala Lys Gly Pro Lys Gly Glu Asn Gly Val
 740 745 750
 Val Gly Pro Thr Gly Pro Val Gly Ala Ala Gly Pro Ala Gly Pro Asn
 755 760 765
 Gly Pro Pro Gly Pro Ala Gly Ser Arg Gly Asp Gly Gly Pro Pro Gly
 770 775 780
 Met Thr Gly Phe Pro Gly Ala Ala Gly Arg Thr Gly Pro Pro Gly Pro
 785 790 795 800
 Ser Gly Ile Ser Gly Pro Pro Gly Pro Pro Gly Pro Ala Gly Lys Glu
 805 810 815
 Gly Leu Arg Gly Pro Arg Gly Asp Gln Gly Pro Val Gly Arg Thr Gly
 820 825 830
 Glu Val Gly Ala Val Gly Pro Pro Gly Phe Ala Gly Glu Lys Gly Pro
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 Ser Gly Glu Ala Gly Thr Ala Gly Pro Pro Gly Thr Pro Gly Pro Gln
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 Gly Leu Leu Gly Ala Pro Gly Ile Leu Gly Leu Pro Gly Ser Arg Gly
 865 870 875 880
 Glu Arg Gly Leu Pro Gly Val Ala Gly Ala Val Gly Glu Pro Gly Pro
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 Leu Gly Ile Ala Gly Pro Pro Gly Ala Arg Gly Pro Pro Gly Ala Val
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 Gly Ser Pro Gly Val Asn Gly Ala Pro Gly Glu Ala Gly Arg Asp Gly
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 Gly Ala Pro Gly Pro His Gly Pro Val Gly Pro Ala Gly Lys His Gly
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 Asn Arg Gly Glu Thr Gly Pro Ser Gly Pro Val Gly Pro Ala Gly Ala
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 Val Gly Pro Arg Gly Pro Ser Gly Pro Gln Gly Ile Arg Gly Asp Lys
 995 1000 1005
 Gly Glu Pro Gly Glu Lys Gly Pro Arg Gly Leu Pro Gly Leu Lys Gly
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 Gln Gly Ala Pro Gly Ser Val Gly Pro Ala Gly Pro Arg Gly Pro Ala
 1045 1050 1055
 Gly Pro Ser Gly Pro Ala Gly Lys Asp Gly Arg Thr Gly His Pro Gly
 1060 1065 1070
 Thr Val Gly Pro Ala Gly Ile Arg Gly Pro Gln Gly His Gln Gly Pro
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 Ala Gly Pro Pro Gly Pro Pro Gly Pro Pro Gly Pro Gly Val Ser
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 Gly Gly Gly Tyr Asp Phe Gly Tyr Asp Gly Asp Phe Tyr Arg Ala Asp
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 1125 1130 1135
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 1235 1240 1245
 Glu Met Ala Thr Gln Leu Ala Phe Met Arg Leu Leu Ala Asn Tyr Ala
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 Ser Gln Asn Ile Thr Tyr His Cys Lys Asn Ser Ile Ala Tyr Met Asp
 1265 1270 1275 1280
 Glu Glu Thr Gly Asn Leu Lys Lys Ala Val Ile Leu Gln Gly Ser Asn
 1285 1290 1295
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 Leu Val Asp Gly Cys Ser Lys Lys Thr Asn Glu Trp Gly Lys Thr Ile
 1315 1320 1325
 Ile Glu Tyr Lys Thr Asn Lys Pro Ser Arg Leu Pro Phe Leu Asp Ile
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<210> 266

<211> 2028

<212> DNA

<213> Homo sapiens

<400> 266

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720
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1920
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2028

<211> 675
 <212> PRT
 <213> Homo sapiens

<400> 267

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Ala Leu Lys Ala Thr His Cys Leu Ala Ala Thr His Trp Ser Pro Ser
 35          40          45
Cys Pro Pro Gln Gln Val Phe Gly Asp Leu Asp Gln Val Arg Met Thr
 50          55          60
Ser Glu Gly Ser Asp Cys Arg Cys Lys Cys Ile Met Arg Pro Leu Ser
 65          70          75
Lys Asp Ala Cys Ser Arg Val Arg Ser Gly Arg Ala Arg Val Glu Asp
 85          90          95
Phe Tyr Thr Val Glu Thr Val Ser Ser Gly Thr Asp Cys Arg Cys Ser
100          105          110
Cys Thr Ala Pro Pro Ser Ser Leu Asn Pro Cys Glu Asn Glu Trp Lys
115          120          125
Met Glu Lys Leu Lys Lys Gln Ala Pro Glu Leu Leu Lys Leu Gln Ser
130          135          140
Met Val Asp Leu Leu Glu Gly Thr Leu Tyr Ser Met Asp Leu Met Lys
145          150          155
Val His Ala Tyr Val His Lys Val Ala Ser Gln Met Asn Thr Leu Glu
165          170          175
Glu Ser Ile Lys Ala Asn Leu Ser Arg Glu Asn Glu Val Val Lys Asp
180          185          190
Ser Val Arg His Leu Ser Glu Gln Leu Arg His Tyr Glu Asn His Ser
195          200          205
Ala Ile Met Leu Gly Ile Lys Lys Glu Leu Ser Arg Leu Gly Leu Gln
210          215          220
Leu Leu Gln Lys Asp Ala Ala Ala Pro Ala Thr Pro Ala Thr Gly
225          230          235
Thr Gly Ser Lys Ala Gln Asp Thr Ala Arg Gly Lys Gly Lys Asp Ile
245          250          255
Ser Lys Tyr Gly Ser Val Gln Lys Ser Phe Ala Asp Arg Gly Leu Pro
260          265          270
Lys Pro Pro Lys Glu Lys Leu Leu Gln Val Glu Lys Leu Arg Lys Glu
275          280          285
Ser Gly Lys Gly Ser Phe Leu Gln Pro Thr Ala Lys Pro Arg Ala Leu
290          295          300
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305          310          315
Lys Gln Glu Val Thr Glu Ala Val Ala Asp Asn Thr Leu Gln Gly Thr
325          330          335
Ser Trp Leu Glu Gln Leu Pro Pro Lys Val Glu Gly Arg Ser Asn Ser
340          345          350
Ala Glu Pro Asn Ser Ala Glu Gln Asp Glu Ala Glu Pro Arg Ser Ser
355          360          365
Glu Arg Val Asp Leu Ala Ser Gly Thr Pro Thr Ser Ile Pro Ala Thr
370          375          380
Thr Thr Thr Ala Thr Thr Thr Pro Thr Pro Thr Thr Ser Leu Leu Pro
385          390          395
Thr Glu Pro Pro Ser Gly Pro Glu Val Ser Ser Gln Gly Arg Glu Ala
405          410          415
Ser Cys Glu Gly Thr Leu Arg Ala Val Asp Pro Pro Val Arg His His
420          425          430
Ser Tyr Gly Arg His Glu Gly Ala Trp Met Lys Asp Pro Ala Ala Arg
435          440          445
Asp Asp Arg Ile Tyr Val Thr Asn Tyr Tyr Tyr Gly Asn Ser Leu Val
450          455          460
Glu Phe Arg Asn Leu Glu Asn Phe Lys Gln Gly Arg Trp Ser Asn Met
465          470          475
Tyr Lys Leu Pro Tyr Asn Trp Ile Gly Thr Gly His Val Val Tyr Gln
480

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	485		490		495
Gly Ala Phe Tyr	Tyr Asn Arg Ala	Phe Thr Lys Asn Ile	Ile Lys Tyr		
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Asp Leu Arg Gln	Arg Phe Val Ala	Ser Trp Ala Leu	Leu Pro Asp Val		
	515		520		525
Val Tyr Glu Asp	Thr Thr Pro Trp	Lys Trp Arg Gly	His Ser Asp Ile		
	530		535		540
Asp Phe Ala Val	Asp Glu Ser Gly	Leu Trp Val Ile	Tyr Pro Ala Val		
	545		550		555
Asp Asp Arg Asp	Glu Ala Gln Pro	Glu Val Ile Val	Leu Ser Arg Leu		
	565		570		575
Asp Pro Gly Asp	Leu Ser Val His	Arg Glu Thr Thr	Trp Lys Thr Arg		
	580		585		590
Leu Arg Arg Asn	Ser Tyr Gly Asn	Cys Phe Leu Val	Cys Gly Ile Leu		
	595		600		605
Tyr Ala Val Asp	Thr Tyr Asn Gln	Gln Glu Gly Gln	Val Ala Tyr Ala		
	610		615		620
Phe Asp Thr His	Thr Gly Thr Asp	Ala Arg Pro Gln	Leu Pro Phe Leu		
	625		630		635
Asn Glu His Ala	Tyr Thr Thr Gln	Ile Asp Tyr Asn	Pro Lys Glu Arg		
	645		650		655
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Phe Val Val					
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<210> 268

<211> 1909

<212> DNA

<213> Homo sapiens

<400> 268

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120
tctactgtat gaattatgct ttaagtagaa ttcaagtcca aggagaactt ggtgaaataa
180
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240
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300
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360
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420
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480
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720
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1020

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 1320
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 1380
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 1800
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<210> 269

<211> 83

<212> PRT

<213> Homo sapiens

<400> 269

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			20				25					30			
Glu	Leu	Gly	Glu	Ile	Asn	Tyr	Phe	Asn	Phe	Phe	Phe	Ile	Leu	Tyr	Lys
		35					40					45			
Ala	Met	Asp	Phe	Ile	Trp	Leu	Met	Cys	Ala	Leu	Tyr	Thr	Ser	His	Phe
		50					55				60				
Asn	Arg	Met	Glu	Leu	Leu	Ile	Ile	Phe	Gln	Arg	Val	Ile	Asp	Met	Gln
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Lys	Phe	Gln													

<210> 270

<211> 1720

<212> DNA

<213> Homo sapiens

<400> 270

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 660
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<210> 271

<211> 256

<212> PRT

<213> Homo sapiens

<400> 271

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				20					25					30	
Leu	Lys	Thr	Leu	Ser	Asn	Gly	Pro	Gln	Ala	Pro	Arg	Arg	Ser	Ala	Pro
				35					40				45		
Leu	Gly	Pro	Val	Ala	Pro	Thr	Arg	Glu	Gly	Val	Glu	Asn	Ala	Cys	Phe
				50					55				60		
Ser	Ser	Glu	Glu	His	Glu	Thr	His	Phe	Gln	Asn	Pro	Gly	Asn	Thr	Arg
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Leu	Gly	Ser	Ser	Pro	Ser	Pro	Pro	Gly	Gly	Val	Ser	Ser	Leu	Pro	Arg

<210> 273
 <211> 284
 <212> FRT
 <213> Homo sapiens

<400> 273
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 35 40 45
 Lys Leu Lys Gly Thr Glu Asp Glu Leu Asp Lys Tyr Ser Glu Ala Leu
 50 55 60
 Lys Asp Ala Gln Glu Lys Leu Glu Leu Ala Glu Lys Lys Ala Thr Asp
 65 70 75 80
 Ala Glu Ala Asp Val Ala Ser Leu Asn Arg Arg Ile Gln Leu Val Glu
 85 90 95
 Glu Glu Leu Asp Arg Ala Gln Glu Arg Leu Ala Thr Ala Leu Gln Lys
 100 105 110
 Leu Glu Glu Ala Glu Lys Ala Ala Asp Glu Ser Glu Arg Gly Met Lys
 115 120 125
 Val Ile Glu Ser Arg Ala Gln Lys Asp Glu Glu Lys Met Glu Ile Gln
 130 135 140
 Glu Ile Gln Leu Lys Lys Glu Ala Lys His Ile Ala Glu Asp Ala Asp Arg
 145 150 155 160
 Lys Tyr Glu Glu Val Ala Arg Lys Leu Val Ile Ile Glu Ser Asp Leu
 165 170 175
 Glu Arg Ala Glu Glu Arg Ala Glu Leu Ser Glu Gly Lys Cys Ala Glu
 180 185 190
 Leu Glu Glu Glu Leu Lys Thr Val Thr Asn Asn Leu Lys Ser Leu Glu
 195 200 205
 Ala Gln Ala Glu Lys Tyr Ser Gln Lys Glu Asp Arg Tyr Glu Glu Glu
 210 215 220
 Ile Lys Val Leu Ser Asp Lys Leu Lys Glu Ala Glu Thr Arg Ala Glu
 225 230 235 240
 Phe Ala Glu Arg Ser Val Thr Lys Leu Glu Lys Ser Ile Asp Asp Leu
 245 250 255
 Glu Asp Glu Leu Tyr Ala Gln Lys Leu Lys Tyr Lys Ala Ile Ser Glu
 260 265 270
 Glu Leu Asp His Ala Leu Asn Asp Met Thr Ser Ile
 275 280

<210> 274
 <211> 2032
 <212> DNA
 <213> Homo sapiens

<400> 274
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<210> 275

<211> 369

<212> PRT

<213> Homo sapiens

<400> 275

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 85 90 95
 Glu Lys Gly Val Lys Leu Lys Leu Thr Ile Val Asp Thr Pro Gly Phe
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 Gly Asp Ala Val Asn Asn Thr Glu Cys Trp Lys Pro Ile Thr Asp Tyr
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 Arg Lys Asn Ile Gln Asp Asn Arg Val His Cys Leu Tyr Phe Ile
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 Ser Pro Phe Gly His Gly Leu Arg Pro Val Asp Val Gly Phe Met Lys
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 Gly Gln Arg Val Arg Gly Arg Leu Tyr Pro Trp Gly Ile Val Glu Val
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<210> 276

<211> 1344

<212> DNA

<213> Homo sapiens

<400> 276

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 <211> 93
 <212> PRT
 <213> Homo sapiens

<400> 277
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 35 40 45
 Ile Met Asp Tyr Tyr Glu Thr Asn Ser Gln Cys Ser Lys Pro Gly Ile
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 Val Phe Ile Thr Lys Arg Gly His Ser Val Cys Thr Asn Pro Ser Asp
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<210> 278
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 <212> DNA
 <213> Homo sapiens

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 Ser Glu Cys Cys Phe Thr Tyr Thr Thr Lys Ile Pro Arg Gln Arg
 35 40 45
 Ile Met Asp Tyr Tyr Glu Thr Asn Ser Gln Cys Ser Lys Pro Gly Ile
 50 55 60
 Val Phe Ile Thr Lys Arg Gly His Ser Val Cys Thr Asn Pro Ser Asp
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<210> 280
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<210> 281
 <211> 93
 <212> PRT
 <213> Homo sapiens

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 Ile Met Asp Tyr Tyr Glu Thr Asn Ser Gln Cys Ser Lys Pro Gly Ile
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<210> 282
 <211> 2750
 <212> DNA
 <213> Homo sapiens

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<210> 283

<211> 380

<212> PRT

<213> Homo sapiens

<220>

<221> VARIANT

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<223> Xaa = Any Amino Acid

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			100					105					110		
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Pro	Asn	Ser	Phe	Leu	Asp	Gln	Glu	Ser	Arg	Arg	Phe	Thr	Ile		
			210			215						220			

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Ala	Arg	Pro	Arg	Gly	His	Gly	Arg	Lys	Gly	Glu	Asp	Ala	Leu	Cys	Arg
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Ser	Gly	Ser	Ser	Ala	Thr	Lys	Ser	Ser	Ser	Thr	Glu	Pro	Ser	Leu	Leu
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<210> 284

<211> 1789

<212> DNA

<213> Homo sapiens

<400> 284

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<211> 335

<212> PRT

<213> Homo sapiens

<400> 285

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 Tyr Glu Arg Ile Asn Lys Ser Met Asn Lys Ser Ile His Ile Val Val
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 Thr Met Ala Lys Ser Leu Glu Asn Ser Val Glu Asn Lys Ile Val Ser
 65 70 75 80
 Leu Asp Pro Ser Glu Ala Gly Pro Pro Arg Tyr Leu Gly Asp Arg Tyr
 85 90 95
 Lys Phe Tyr Leu Glu Asn Leu Thr Leu Gly Ile Arg Glu Ser Arg Lys
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 Glu Asp Glu Gly Trp Tyr Leu Met Thr Leu Glu Lys Asn Val Ser Val
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 Gln Arg Phe Cys Leu Gln Leu Arg Leu Tyr Glu Gln Val Ser Thr Pro
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 Glu Ile Lys Val Leu Asn Lys Thr Gln Glu Asn Gly Thr Cys Thr Leu
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 Ser Glu Lys Ala Gly Thr His Pro Leu Asn Pro Ala Asn Ser Ser His
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 Leu Leu Ser Leu Thr Leu Gly Pro Gln His Ala Asp Asn Ile Tyr Ile
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 Cys Thr Val Ser Asn Pro Ile Ser Asn Asn Ser Gln Thr Phe Ser Pro
 210 215 220
 Trp Pro Gly Cys Arg Thr Asp Pro Ser Glu Thr Lys Pro Trp Ala Val
 225 230 235 240
 Tyr Ala Gly Leu Leu Gly Gly Val Ile Met Ile Leu Ile Met Val Val
 245 250 255
 Ile Leu Gln Leu Arg Arg Arg Gly Lys Thr Asn His Tyr Gln Thr Thr
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 Val Glu Lys Lys Ser Leu Thr Ile Tyr Ala Gln Val Gln Lys Pro Gly
 275 280 285
 Pro Leu Gln Lys Lys Leu Asp Ser Phe Pro Ala Gln Asp Pro Cys Thr

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Thr	Ile	Tyr	Val	Ala	Ala	Thr	Glu	Pro	Val	Pro	Glu	Ser	Val	Gln	Glu
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 <212> PRT
 <213> Homo sapiens

<400> 286															
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Pro	Lys	Ile	Leu	Arg	Gln	Leu	Gly	Ser	Lys	Val	Leu	Leu	Pro	Leu	Thr
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Tyr	Glu	Arg	Ile	Asn	Lys	Ser	Met	Asn	Lys	Ser	Ile	His	Ile	Val	Val
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Thr	Met	Ala	Lys	Ser	Leu	Glu	Asn	Ser	Val	Glu	Asn	Lys	Ile	Val	Ser
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Leu	Asp	Pro	Ser	Glu	Ala	Gly	Pro	Pro	Arg	Tyr	Leu	Gly	Asp	Arg	Tyr
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Lys	Phe	Tyr	Leu	Glu	Asn	Leu	Thr	Leu	Gly	Ile	Arg	Glu	Ser	Arg	Lys
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Glu	Asp	Glu	Gly	Trp	Tyr	Leu	Met	Thr	Leu	Glu	Lys	Asn	Val	Ser	Val
		115				120						125			
Gln	Arg	Phe	Cys	Leu	Gln	Leu	Arg	Leu	Tyr	Glu	Gln	Val	Ser	Thr	Pro
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Glu	Ile	Lys	Val	Leu	Asn	Lys	Thr	Gln	Glu	Asn	Gly	Thr	Cys	Thr	Leu
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Ile	Leu	Gly	Cys	Thr	Val	Glu	Lys	Gly	Asp	His	Val	Ala	Tyr	Ser	Trp
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Ser	Glu	Lys	Ala	Gly	Thr	His	Pro	Leu	Asn	Pro	Ala	Asn	Ser	Ser	His
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Leu	Leu	Ser	Leu	Thr	Leu	Gly	Pro	Gln	His	Ala	Asp	Asn	Ile	Tyr	Ile
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Cys	Thr	Val	Ser	Asn	Pro	Ile	Ser	Asn	Asn	Ser	Gln	Thr	Phe	Ser	Pro
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Trp	Pro	Gly	Cys	Arg	Thr	Asp	Pro	Ser	Gly	Lys	Thr	Asn	His	Tyr	Gln
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Thr	Thr	Val	Glu	Lys	Lys	Ser	Leu	Thr	Ile	Tyr	Ala	Gln	Val	Gln	Lys
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Pro	Gly	Pro	Leu	Gln	Lys	Lys	Leu	Asp	Ser	Phe	Pro	Ala	Gln	Asp	Pro
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Cys	Thr	Thr	Ile	Tyr	Val	Ala	Ala	Thr	Glu	Pro	Val	Pro	Glu	Ser	Val
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Gln	Glu	Thr	Asn	Ser	Ile	Thr	Val	Tyr	Ala	Ser	Val	Thr	Leu	Pro	Glu
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Ser
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 <212> PRT
 <213> Homo sapiens

<400> 287															
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		20						25					30		
Pro	Lys	Ile	Leu	Arg	Gln	Leu	Gly	Ser	Lys	Val	Leu	Leu	Pro	Leu	Thr
		35				40					45				
Tyr	Glu	Arg	Ile	Asn	Lys	Ser	Met	Asn	Lys	Ser	Ile	His	Ile	Val	Val

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Thr Met Ala Lys Ser Leu	Glu Asn Ser Val	Glu Asn Lys Ile Val Ser
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Leu Asp Pro Ser Glu Ala Gly	Pro Pro Arg Tyr	Leu Gly Asp Arg Tyr
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Lys Phe Tyr Leu Glu Asn Leu Thr	Leu Gly Ile Arg Glu Ser Arg Lys	
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Glu Asp Glu Gly Trp Tyr Leu Met Thr	Leu Glu Lys Asn Val Ser Val	
115	120	125
Gln Arg Phe Cys Leu Gln Leu Arg Leu Tyr	Glu Gln Val Ser Thr Pro	
130	135	140
Glu Ile Lys Val Leu Asn Lys Thr Gln Glu Asn Gly	Thr Cys Thr Leu	
145	150	155
Ile Leu Gly Cys Thr Val Glu Lys Gly Asp His Val Ala Tyr Ser Thr		
165	170	175
Ser Glu Lys Ala Gly Thr His Pro Leu Asn Pro Ala Asn Ser Ser His		
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Leu Leu Ser Leu Thr Leu Gly Pro Gln His Ala Asp Asn Ile Tyr Ile		
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Cys Thr Val Ser Asn Pro Ile Ser Asn Asn Ser Gln Thr Phe Ser Pro		
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Trp Pro Gly Cys Arg Thr Asp Pro Ser Glu Thr Lys Pro Trp Ala Val		
225	230	235
Tyr Ala Gly Leu Leu Gly Gly Val Ile Met Ile Leu Ile Met Val Val		
245	250	255
Ile Leu Gln Leu Arg Arg Arg Gly Lys Thr Asn His Tyr Gln Thr Thr		
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Val Glu Lys Lys Ser Leu Thr Ile Tyr Ala Gln Val Gln Lys Pro Gly		
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<211> 3640

<212> DNA

<213> Homo sapiens

<400> 288

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<210> 289

<211> 628

<212> PRT

<213> Homo sapiens

<400> 289

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 35 40 45
 Ala Asp Thr Thr Cys Gly Gln Asn Ala Thr Glu Leu Tyr Cys Phe Tyr
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 Ser Glu Asn Thr Asp Leu Thr Cys Arg Gln Pro Lys Cys Asp Lys Cys
 65 70 75 80
 Asn Ala Ala Tyr Pro His Leu Ala His Leu Pro Ser Ala Met Ala Asp
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 Ser Ser Phe Arg Phe Pro Arg Thr Trp Trp Gln Ser Ala Glu Asp Val
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 His Arg Glu Lys Ile Gln Leu Asp Leu Glu Ala Glu Phe Tyr Phe Thr
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 His Leu Ile Val Met Phe Lys Ser Pro Arg Pro Ala Ala Met Val Leu
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 Ala Thr Asn Cys Ser Ala Thr Phe Gly Leu Glu Asp Asp Val Val Lys
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 Gly Gly Glu Val Ile Phe Lys Ala Leu Ser Pro Pro Tyr Asp Thr Glu
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 225 230 235 240
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 His Trp Tyr His Asn Gly Ala Pro Met Glu Ser Asp Gly Gln Ala Gly
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 Pro Arg Glu Ser Pro Ala Gln Val Leu Lys Pro Gly Lys Thr Gln Leu
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 Val Tyr Gly Pro Gly Glu Lys Gln Ser Gln Asp Leu Trp Val Asp Leu
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 180 185 190
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 225 230 235 240
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 Ser Lys Ile Thr Thr Thr Ser Ala Val Glu Phe Thr Pro Leu Pro Thr
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His	His	Trp	Pro	Ala	Met	Lys	Phe	His	Asn	His	Pro	Asn
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<212> DNA

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 Trp Asn Glu Glu Gly Val Glu Val Asp Ser Gln Ala Tyr Asn His Arg
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 65 70 75 80
 Ser Met Gly Gln Ala Ser Pro Glu Ser Lys Gly Phe Thr Asp Leu Leu
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 His Asn Tyr Tyr Ile Ser Arg Ile Tyr Gly Pro Ala Asp Ser Ala Ser
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 Arg Asp Leu Trp Val Asn Ile Asp Gln Met Glu Lys Asp Lys Val Lys
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 Ile His Gly Ile Leu Ser Asn Thr His Arg Gln Ala Ala Arg Val Asn
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 Phe Gly Tyr Lys Glu Ile Pro Val Leu Val Thr Gln Ile Ser Ser Thr
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 385 390 395 400
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 Pro Phe Cys Asn Ala Ser Leu Pro Ala Gln Arg Trp Lys Trp Val Ser
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 Arg Asn Arg Leu Phe Asn Leu Gly Ala Thr Gln Cys Leu Gly Thr Gly
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 Trp Pro Val Thr Asn Thr Thr Val Ser Leu Gly Met Tyr Glu Cys Asp
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 Arg Glu Ala Leu Ser Leu Arg Met Ala Val Ser Tyr Thr Arg Gly Pro
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 Val Val Pro Ala Ser Gly Gly Ser Cys Lys Gln Cys Ile Gln Ala Trp
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 Gly Ser Glu Glu Asp Leu Cys Ala Arg Pro Tyr Tyr Glu Val Tyr Thr
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 195 200 205
 His Leu Trp Cys Ala Thr Thr Gln Asp Tyr Gly Lys Asp Glu Arg Trp
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 Gly Phe Cys Pro Ile Lys Ser Asn Asp Cys Glu Thr Phe Trp Asp Lys
 225 230 235 240
 Asp Gln Leu Thr Asp Ser Cys Tyr Gln Phe Asn Phe Gln Ser Thr Leu
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 325 330 335
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 405 410 415
 Leu Ser Ile His Ser Met Ala Glu Leu Glu Phe Ile Thr Lys Gln Ile
 420 425 430
 Lys Gln Glu Val Glu Glu Leu Trp Ile Gly Leu Asn Asp Leu Lys Leu
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 Gln Met Asn Phe Glu Trp Ser Asp Gly Ser Leu Val Ser Phe Thr His
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 Trp His Pro Phe Glu Pro Asn Asn Phe Arg Asp Ser Leu Glu Asp Cys
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 Val Thr Ile Trp Gly Pro Glu Gly Arg Trp Asn Asp Ser Pro Cys Asn
 485 490 495
 Gln Ser Leu Pro Ser Ile Cys Lys Lys Ala Gly Arg Leu Ser Gln Gly
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 Glu Tyr Phe Trp Thr Ala Leu Gln Asp Leu Asn Ser Thr Gly Ser Phe
 Arg Trp Leu Ser Gly Asp Glu Val Ile Tyr Thr His Trp Asn Arg Asp
 Gln Pro Gly Tyr Arg Arg Gly Gly Cys Val Ala Leu Ala Thr Gly Ser
 Ala Met Gly Leu Trp Glu Val Lys Asn Cys Thr Ser Phe Arg Ala Arg
 Tyr Ile Cys Arg Gln Ser Leu Gly Thr Pro Val Thr Pro Glu Leu Pro
 Gly Pro Asp Pro Thr Pro Ser Leu Thr Gly Ser Cys Pro Gln Gly Trp
 Val Ser Asp Pro Lys Leu Arg His Cys Tyr Lys Val Phe Ser Ser Glu
 Arg Leu Gln Glu Lys Lys Ser Trp Ile Gln Ala Leu Gly Val Cys Arg
 Glu Leu Gly Ala Gln Leu Leu Ser Leu Ala Ser Tyr Glu Glu Glu His
 Phe Val Ala His Met Leu Asn Lys Ile Phe Gly Glu Ser Glu Pro Glu
 Ser His Glu Gln His Trp Phe Trp Ile Gly Leu Asn Arg Arg Asp Pro
 Arg Glu Gly His Ser Trp Arg Trp Ser Asp Gly Leu Gly Phe Ser Tyr
 His Asn Phe Ala Arg Ser Arg His Asp Asp Asp Ile Arg Gly Cys
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 Thr Gln Leu Asp Trp Ile Cys Lys Ile Pro Arg Gly Val Asp Val Arg
 Glu Pro Asp Ile Gly Arg Gln Gly Arg Leu Glu Trp Val Arg Phe Gln
 Glu Ala Glu Tyr Lys Phe Phe Glu His His Ser Ser Trp Ala Gln Ala
 Gln Arg Ile Cys Thr Trp Phe Gln Ala Asp Leu Thr Ser Val His Ser
 Gln Ala Glu Leu Gly Phe Leu Gly Gln Asn Leu Gln Lys Leu Ser Ser
 Asp Gln Glu Gln His Trp Trp Ile Gly Leu His Thr Leu Gln Ser Asp
 Gly Arg Phe Arg Trp Thr Asp Gly Ser Ile Ile Asn Phe Ile Ser Trp
 Ala Pro Gly Lys Pro Arg Pro Ile Gly Lys Asp Lys Cys Val Tyr
 Met Thr Ala Arg Gln Glu Asp Trp Gly Asp Gln Arg Cys His Thr Ala
 Leu Pro Tyr Ile Cys Lys Arg Ser Asn Ser Ser Gly Glu Thr Gln Pro
 Gln Asp Leu Pro Pro Ser Ala Leu Gly Gly Cys Pro Ser Gly Trp Asn
 Gln Phe Leu Asn Lys Cys Phe Arg Ile Gln Gly Gln Asp Pro Gln Asp
 Arg Val Lys Trp Ser Glu Ala Gln Phe Ser Cys Glu Gln Gln Glu Ala
 Gln Leu Val Thr Ile Ala Asn Pro Leu Glu Gln Ala Phe Ile Thr Ala
 Ser Leu Pro Asn Val Thr Phe Asp Leu Trp Ile Gly Leu His Ala Ser
 Gln Arg Asp Phe Gln Trp Ile Glu Gln Glu Pro Leu Leu Tyr Thr Asn
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 Lys Pro Thr Ser Cys Ala Val Ile Leu His Ser Pro Ser Ala His Phe

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 <212> FRT
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<400> 307

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 Tyr Thr Val Gln Gln Tyr Glu Asn Glu Glu Gly Lys Trp Val Leu Ile
 50 55 60
 Gly Ser Pro Leu Val Gly Gln Pro Lys Ala Arg Thr Gly Asp Val Tyr
 65 70 75 80
 Lys Cys Pro Val Gly Arg Glu Arg Ala Met Pro Cys Val Lys Leu Asp
 85 90 95
 Leu Pro Val Asn Thr Ser Ile Pro Asn Val Thr Glu Ile Lys Glu Asn
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 Met Thr Phe Gly Ser Thr Leu Val Thr Asn Pro Asn Gly Gly Phe Leu
 115 120 125
 Ala Cys Gly Pro Leu Tyr Ala Tyr Arg Cys Gly His Leu His Tyr Thr
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 Thr Gly Ile Cys Ser Asp Val Ser Pro Thr Phe Gln Val Val Asn Ser
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 Phe Ala Pro Val Gln Glu Cys Ser Thr Gln Leu Asp Ile Val Ile Val
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 Leu Asp Gly Ser Asn Ser Ile Tyr Pro Trp Glu Ser Val Ile Ala Phe
 180 185 190
 Leu Asn Asp Leu Leu Lys Arg Met Asp Ile Gly Pro Lys Gln Thr Gln
 195 200 205
 Val Gly Ile Val Gln Tyr Gly Glu Asn Val Thr His Glu Phe Asn Leu
 210 215 220
 Asn Lys Tyr Ser Ser Thr Glu Glu Val Leu Val Ala Ala Asn Lys Ile
 225 230 235 240
 Gly Arg Gln Gly Gly Leu Gln Thr Met Thr Ala Leu Gly Ile Asp Thr
 245 250 255
 Ala Arg Lys Glu Ala Phe Thr Glu Ala Arg Gly Ala Arg Gly Val
 260 265 270
 Lys Lys Val Met Val Ile Val Thr Asp Gly Glu Ser His Asp Asn Tyr
 275 280 285
 Arg Leu Lys Gln Val Ile Gln Asp Cys Glu Asp Glu Asn Ile Gln Arg
 290 295 300
 Phe Ser Ile Ala Ile Leu Gly His Tyr Asn Arg Gly Asn Leu Ser Thr
 305 310 315 320
 Glu Lys Phe Val Glu Glu Ile Lys Ser Ile Ala Ser Glu Pro Thr Glu
 325 330 335
 Lys His Phe Phe Asn Val Ser Asp Glu Leu Ala Leu Val Thr Ile Val
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 Lys Ala Leu Gly Glu Arg Ile Phe Ala Leu Glu Ala Thr Ala Asp Gln
 355 360 365
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 370 375 380
 His Tyr Ser Gln Asp Trp Val Met Leu Gly Ala Val Gly Ala Tyr Asp
 385 390 395 400
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 405 410 415
 His Asn Thr Thr Phe Gln Thr Glu Pro Ala Lys Met Asn Glu Pro Leu
 420 425 430
 Ala Ser Tyr Leu Gly Tyr Thr Val Asn Ser Ala Thr Ile Pro Gly Asp
 435 440 445
 Val Leu Tyr Ile Ala Gly Gln Pro Arg Tyr Asn His Thr Gly Gln Val
 450 455 460
 Val Ile Tyr Lys Met Glu Asp Gly Asn Ile Asn Ile Leu Gln Thr Leu
 465 470 475 480
 Gly Gly Glu Gln Ile Gly Ser Tyr Phe Gly Ser Val Leu Thr Thr Ile
 485 490 495
 Asp Ile Asp Lys Asp Ser Tyr Thr Asp Leu Leu Leu Val Gly Ala Pro
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 Met Tyr Met Gly Thr Glu Lys Glu Glu Gln Gly Lys Val Tyr Val Tyr

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 Arg Gln Thr Cys Cys Ser Ser Leu Lys Asp Asn Ser Cys Thr Lys Glu
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 Asn Lys Asn Glu Pro Cys Gly Ala Arg Phe Gly Thr Ala Ile Ala Ala
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 Val Lys Asp Leu Asn Val Asp Gly Phe Asn Asp Val Val Ile Gly Ala
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 Pro Leu Glu Asp Asp His Ala Gly Ala Val Tyr Ile Tyr His Gly Ser Ser
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 Gly Lys Thr Ile Arg Glu Ala Tyr Ala Gln Arg Ile Pro Ser Gly Gly
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 Asp Gly Lys Thr Leu Lys Phe Phe Gly Gln Ser Ile His Gly Glu Met
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 Asp Leu Asn Gly Asp Gly Leu Thr Asp Val Thr Ile Gly Gly Leu Gly
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 Met Asn Phe Glu Pro Asn Lys Val Asn Ile Gln Lys Lys Asn Cys Arg
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 Val Glu Gly Lys Glu Thr Val Cys Ile Asn Ala Thr Met Cys Phe His
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 Ile Pro Phe Ala Lys Asp Cys Gly Asn Lys Glu Arg Cys Ile Ser Asp
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 930
 Tyr Glu Val Gly Leu Gln Phe Tyr Ser Ser Ala Ser Glu His His Ile
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 Ser Val Ala Ala Asn Glu Thr Ile Pro Glu Phe Ile Asn Ser Thr Glu
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Leu Trp Lys Pro Thr Phe Ile Arg Ala His Phe Ser Ser Leu Asn Leu					
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Thr Leu Arg Gly Glu Leu Lys Ser Glu Asn Ser Ser Leu Thr Leu Ser					
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Ser Ser Asn Arg Lys Arg Glu Leu Ala Ile Gln Ile Ser Lys Asp Gly					
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Leu Pro Gly Arg Val Pro Leu Trp Val Ile Leu Leu Ser Ala Phe Ala					
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Gly Leu Leu Leu Leu Met Leu Leu Ile Leu Ala Leu Trp Lys Ile Gly					
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2383

PEM's
con plate with table # 25 (pem3) + # 47 (pem6) are G1, rest are G3

Table 1. Previously characterized and novel Pan Endothelial Markers. The most abundant tags derived by summing the tags from Normal EC (N-EC's) and Tumor EC (T-EC's) SAGE libraries are listed in descending order. N-EC and T-EC SAGE libraries contained 98,694 and 98,688 SAGE tags respectively. For comparison, the corresponding number of SAGE tags found in cultured human umbilical vein endothelial cells (HUV-EC), human dermal microvascular endothelial cells (HMVEC), and non-endothelial cell lines (Cell Lines) are shown. The HMVEC SAGE library contained 280,000 tags and the HMVEC library 111,000 tags. Non-endothelial cell lines consisted of 1.8x10⁶ tags derived from a total of 14 different cancer cell lines including colon, breast, lung, and pancreatic cancers, as well as one non-transformed keratinocyte cell line, two kidney epithelial cell lines, and normal monocytes. Tag numbers for each group were normalized to 100,000 transcripts. A description of the gene product corresponding to each tag is given, followed by alternative names in parenthesis. The sequence CATG precedes all tags and the 15th base (11th shown) was determined as previously described by Velculescu et al. (Nat. Genet. 1999 Dec;23(4):387-8).

no.	Tag Sequence	N-EC's	T-EC's	HUVEC	HMVEC	Cell Lines	Description
1	CATATCAITAA	247	501	130	87	2	angiomodulin (ANG), IGFBP-7, IGFBP-rP1, Mac25, TAF
2	TGCACITCAAG	328	141	0	0	0	helix
3	TTTGCACTTT	185	84	181	115	4	connective tissue growth factor (CTGF, IGFBP-rP2)
4	CCCTGTCCG	131	104	1	1	0	ESTs
5	TTGTGACTT	73	131	2	14	1	collagen, type VI, alpha 1
6	ACCATGGATT	102	87	0	0	2	interferon induced transmembrane protein 1 (S-27, Leu 13)
7	ACACTCTTTC	104	44	60	62	2	guanine nucleotide binding protein 11
8	TTCTGCTTTC	71	67	118	72	0	von Willebrand factor
9	TCCGTGGCAGA	68	88	3	13	3	cysteine-rich protein 2 (CRP-2, ESP-1, SmlLM)
10	TATCTCTCAAG	26	106	34	18	1	collagen, type XVIII, alpha 1
11	ATGCTCTTCT	58	65	17	17	3	insulin-like growth factor-binding protein 4
12	GGGATTAAGC	40	67	30	14	2	CD148 (S-Endo 1, P1H12, Muc18, MCAM, Mel-CAM)
13	TAGTGTGTA	38	69	9	13	0	collagen, type IV, alpha 2
14	TTCTCCCAAT	20	85	16	64	2	SPARC (osteonectin, BM-40)
15	GTGTCAGCG	24	74	0	10	2	collagen, type IV, alpha 2
16	GTTTATGATA	35	56	11	11	1	matrix Gla protein (MGP)
17	CCCTTTCAC	52	33	0	0	0	ESTs, weakly similar to HPERII-7 protein
18	TTCTGTGGAG	58	27	18	56	2	gap junction protein, alpha 1, 43kD (connexin 43)
19	AAGATCAAGT	34	50	2	4	1	actin, alpha 1, skeletal muscle / actin, alpha 2, smooth muscle, aorta
20	TCTGTGAGCAT	32	48	0	0	0	small inducible cytokine subfamily B (Cys-X-Cys), member 14 (BRAV)
21	CAGGTTTCATA	22	56	0	0	0	aggrucanase 1 (metalloproteinase with thrombospondin type 1 motifs, 4)
22	GCACAAGTTGT	43	25	6	22	0	calcitonin receptor-like receptor activity modifying protein 2
23	AGCTGTGGCC	45	23	0	0	0	calcitonin receptor-like receptor activity modifying protein 3
24	CTTGTGGATA	13	54	12	0	0	cell division cycle 42 (GTP-binding protein, 25kD)
25	CAACAATAA	42	25	13	6	0	ESTs

26	ACCGGGCCCG	50	15	0	0	0	0	telranectin (plasminogen-binding protein)
27	GAAGCTAAGT	35	27	0	5	1	0	osteoblast specific factor 2 (ascidin-like)
28	GAAATTTAACC	38	21	0	3	0	0	solute carrier family 21 (prosligand transporter), member 2
29	GATAACTAAGT	18	35	4	4	0	0	angiomodulin (ANG, IGFBP-7, IGFBP-P1, Mac25, TAF)
30	TATGAGGGTAA	19	30	40	2	0	0	regulator of G-protein signalling 5
31	CCACGGGATTC	10	39	0	0	0	0	collagen, type III, alpha 1(I)
32	TTTACAAGAG	26	21	0	1	1	1	carboxypeptidase E
33	CCAGCTAAGAT	22	25	0	16	1	0	cysteine and glycine-rich protein 2 (LIM domain only, smooth muscle)
34	ACAAAGCATTT	26	20	0	14	1	0	Human insulin-like growth factor binding protein 5 (IGFBP5) mRNA
35	GCCTGTCGCTC	8	38	22	11	0	0	ESTs / biglycan
36	TACITTTATAAG	25	21	1	1	0	0	metalloproteinase with thrombospondin type 1 motifs (ADAMTS1, METH-1)
37	TGTTTATAGA	15	29	21	1	1	0	ESTs / erythrocyte membrane protein band 4.1-like 2
38	GTCCCTGCCTT	18	25	1	1	0	0	glutathione S-transferase M2 (muscle)
39	GAGCCTCATTA	21	21	2	2	1	0	ESTs / GTP-binding protein overexpressed in skeletal muscle
40	GGCCTACAGT	26	13	2	3	0	0	ESTs / KIAA0321 protein
41	GCTAACCCCTG	7	31	0	1	0	0	ESTs
42	ATCACACAGGT	19	18	0	0	0	0	thyroid and eye muscle autoantigen D1 (64KD)
43	ACAAGTACTGT	18	19	36	27	0	0	cadherin 5, VE-cadherin (vascular epithelium)
44	TACCGTGGAC	20	17	0	1	0	0	selectin P (granule membrane protein 140KD, antigen CD62)
45	ACATTCGAAT	18	18	0	1	1	0	tissue inhibitor of metalloproteinase 3
46	GAGCTGGGATA	6	29	0	0	0	0	chondroin sulfate proteoglycan 4 (melanoma-associated)
47	GGCACTCCTGT	22	13	19	12	0	0	ESTs
48	TACAGGCCCC	20	15	8	5	0	0	ESTs
49	TGGCAGGTGCA	10	23	0	1	0	0	albumin
50	TGGGAACCTG	11	22	0	1	1	0	eukaryotic translation initiation factor 4 gamma, 1
51	TTTATCCACT	20	13	0	2	0	0	ESTs, KIAA0362 protein
52	AACAGGGGCCA	15	18	0	0	1	0	Interferon, alpha-inducible protein (clone IFI-6-16)
53	ACTGAAGAAG	6	26	0	0	1	0	complement component 1, s subcomponent
54	ACCGTCTCTGA	8	24	10	6	0	0	transcription factor 4
55	ATACTATAATT	25	6	12	0	0	0	ESTs
56	TTTGTATAGAA	17	15	4	5	1	0	hct domain and RLD 2
57	GTAAATGACAGA	20	11	1	1	1	1	slaninocalcin
58	ATAGGGGAAA	13	19	4	1	0	0	ESTs, KIAA1075 protein
59	GTGCTACTTCT	5	25	2	18	0	0	collagen, type IV, alpha 1
60	CCGCGCCCTCC	6	24	0	0	1	0	peanut (Drosophila)-like 2
61	TTGAATTTGT	19	10	1	1	0	0	RNA-binding protein gene with multiple splicing
62	CGAGAGTGTGA	22	6	0	0	0	0	ESTs
63	CCCTGTTTCAAG	14	15	38	24	0	0	tyrosine kinase with IgG and EGF homology domains (Tie)

64	CAGATGGAGGC	18	10	1	9	0	ESTs
65	AGGCTCTGGC	8	20	0	0	0	ESTs
66	TGCTGTTAG	20	8	40	15	0	ESTs
67	GGCTTAGGATG	18	9	10	14	0	ESTs
68	GGTTGTCGG	6	21	0	0	1	ESTs
69	ACAAGTACCCA	5	22	4	5	0	P311 protein
70	CTTCTCTTGAG	18	9	1	4	1	basic transcription element binding protein 1
71	GCTAATAATGT	10	17	0	2	0	KIAA1077 protein
72	TGTGCTTTT	10	15	1	4	0	KIAA0758 protein / protein kinase, cAMP-dependent, catalytic, alpha
73	CATGACGGATC	17	8	0	1	0	interleukin 1 receptor, type 1
74	GCAGCAGCAGC	6	18	0	2	0	T-box 2
75	TGACTGTATTA	13	11	0	0	0	ESTs / amine oxidase, copper containing 3 (vascular adhesion protein 1)
76	GAATGCTCTTG	6	18	0	11	0	gap junction protein, alpha 4, 37kD (connexin 37)
77	GTAGTTCGTGA	18	6	0	5	0	ESTs, clone 23698 mRNA
78	TCCCTCTCTC	6	17	0	0	0	periodontal ligament fibroblast protein
79	GGCAGTGGCT	5	18	12	5	0	ESTs, DKFZP588B0621 protein
80	AAATATGTGT	18	4	13	3	0	ESTs
81	GTCATTTTCTA	11	11	10	2	0	transcription factor 8 (represses interleukin 2 expression)
82	CTCTCGAAACC	14	8	0	0	0	complement component 1 inhibitor (angiogenesis, hereditary)
83	TAAATGTGTAA	4	18	0	0	0	guanylate cyclase 1, soluble, beta 3
84	TCAAGCAATCA	13	9	0	1	0	ESTs
85	GAAGACACTTG	15	7	1	0	0	ESTs
86	GGGTAGGGTGA	6	15	0	0	1	Integrin, alpha 7
87	TGSAACAGTGA	10	10	10	5	0	ESTs
88	GAGTGGGTACC	10	9	0	1	0	ESTs
89	GTGAGGGTCCC	13	7	0	1	0	decidual protein induced by progesterone
90	GTCACTCACTT	14	8	4	1	0	halcy (Drosophila)-homolog
91	AGCAGAGACAA	14	8	1	10	0	neuropeptide receptor A - guanylate cyclase A
92	AGCGATGGAGA	9	10	0	0	0	ESTs
93	CGTGGGGTGTA	9	10	17	3	0	

TEM's complete web table

Table 2. SAGE tags elevated in tumor endothelium. The top 46 tags with the highest tumor EC (T-ECs) to normal EC (NECs) tag ratios are listed in descending order. To calculate tag ratios, a value of 0.5 was assigned in cases where zero tags were observed. The SAGE libraries are the same as those listed in Table 1. Tag numbers for each group were normalized to 100,000 transcripts. A 'Description' of the gene product corresponding to each tag is given, followed by alternative names in parentheses. †: multiple tags for this gene are due to alternative polyadenylation sites.

no.	Tag Sequence	NECs	T-ECs	HUVEC	HMVEC	Cell Lines	Description
1	GGGGTGGCCA	0	28	0	2	0	ESTs, similarity to thrombospondin
2	GATCTCGGTGT	0	25	0	0	0	ESTs, similarity to rat Rhes ras-related protein
3	CAITTTATCT	0	23	1	0	0	ESTs
4	CTTCTTTTGG	0	22	6	20	1	regulated in glioma-like 7-1 (DKK-3/REIC)
5	TATTAAGTCTC	0	21	1	3	1	ESTs, similarity to JNK Interacting protein-3a
6	CAGGAGACCC	0	18	2	0	0	MMP-11 (stromelysin 3)
7	GGAATGTCAA	1	31	53	22	1	MMP-2 (gelatinase A, 72KD type IV collagenase)
8	CCTGGTTCAGT	0	15	0	0	0	ESTs
9	TTTTTAAGAAC	0	14	1	4	0	ESTs
10	TTTGGTTTCC	5	139	1	16	0	collagen, type I, alpha 2, transcript A ¹
11	ATTTGTATGA	0	13	4	8	0	nidogen (entactin)
12	ACTTTAGATGG	1	23	0	15	0	collagen, type VI, alpha 3
13	GAGTGAGACCC	3	63	0	0	1	Thy-1 cell surface antigen
14	GTACACACCC	0	10	0	0	0	ESTs / cytostatin S
15	CCACAGGGGAT	2	38	0	2	1	collagen, type III, alpha 1
16	TTAAAGTCA	1	19	1	3	1	ESTs
17	ACAGACTGTTA	4	74	0	0	0	ESTs, similarity with sea squirt nidogen
18	CCACTGCAACG	1	18	1	1	0	ESTs, similarity with homeobox protein DLX-3
19	GTTCACAGAA	0	9	0	3	0	collagen, type I, alpha 2, transcript B ¹
20	TACCACTGCC	0	9	4	1	1	ESTs / pregnancy specific beta-1-glycoprotein 1
21	GCCCTTTCTGT	1	17	3	1	2	endo180 lectin
22	TTAAATGAGC	2	33	0	4	0	collagen, type I, alpha 1
23	AGACATACTGA	1	16	1	0	0	ESTs, DKGZP434G162 protein
24	TCGCCACAGGAG	1	16	0	0	0	bone morphogenetic protein 1 (metalloproteinase)
25	AGCCCAAGTG	0	8	0	0	0	sllt (Drosophila) homolog 3 (MEGFS)
26	ACTAGCAATAC	0	8	0	0	0	KIAA0672 gene product
27	TACAAATCGTT	0	8	0	0	0	

See table 2 from paper for Gly2dr G3
 all
 G3

28	TTGGGTGAAA	0	8	0	0	0	ESTs
30	CATTATCGAAA	0	8	0	0	0	Integrin, alpha 1
31	AGAAACACGG	0	8	2	7	0	collagen, type IV, alpha 1
32	ACCAAAACAC	0	8	0	3	0	
33	TGAAATAAAC	0	8	3	1	1	
34	TTGGTTTCC	1	15	0	0	0	ESTs
35	GTGGAGACGGA	1	15	1	2	1	ESTs
36	TTTGTGTTGTA	1	14	2	0	0	collagen, type XII, alpha 1
37	TTATGTTTAAT	3	39	0	0	1	lumican
38	TGGAATGACC	15	179	0	40	0	ESTs / collagen, type I, alpha 1
39	TGCCACACAGT	1	13	0	2	0	transforming growth factor, beta 3
40	GATGAGGAGAC	3	35	0	18	1	collagen, type I, alpha 2, transcript C
41	ATCAAGGTTT	2	23	0	0	0	ESTs, DKFZp564O222 mRNA
42	AGTCACATGT	1	11	2	0	0	cell division cycle 42 (GTP-binding protein)
43	TTGGTTGGTC	4	45	0	19	0	
44	CCCCACCGGG	2	21	0	0	0	ESTs
45	GGCTTGCCTTT	1	10	0	10	1	
46	ATCCGTTCCCG	1	10	1	0	0	peanut-like protein 1

Table 3. Detection of transcripts in various tumor types by RT-PCR and in situ hybridization (ISH). The "+" sign indicates the presence of a robust RT-PCR product or strong positive staining of vessels by in situ hybridization. The "-" sign indicates an undetectable signal by in situ hybridization or an absent or barely detectable transcript by RT-PCR. The "+/-" sign indicates a very weak signal in a limited number of vessels by in situ hybridization.

	TEM1	TEM3	TEM4	TEM5	TEM7	TEM8	TEM9	vWF	Hevin
RT-PCR									
Colon Nor.	+	+	+	+	+	+	+	+	ND
Colon Tum.	+	+	+	+	+	+	+	+	ND
Colon Nor.	+	+	+	+	+	+	+	+	+
Colon Tum.	+	+	+	+	+	+	+	+	+
Liver Met.	+	+/-	+	+	+	+	+	+/-	ND
Lung Tum.	+	ND	+	+	+	+	+	+	+
Brain Tum.	+	ND	ND	ND	+	ND	ND	+	+
Corpus Lut.	+	+	+	+	+	+	+	+	+
Wound	+	ND	+	ND	+/-	+/-	ND	+	+

* hevin was localized to both endothelial cells and malignant cells in brain tissue.
 ND: not determined.

www.sagenet.org/angletable3.htm (to be posted upon publication)

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Table 3. SAGE tags elevated in normal endothelium. The top 46 tags with the highest normal EC (N-EC's) to tumor EC (T-EC's) tag ratios are listed in descending order. To calculate tag ratios, a value of 0.5 was assigned in cases where zero tags were observed. The SAGE libraries are the same as those listed in Table 1. Tag numbers for each group were normalized to 100,000 transcripts. A 'Description' of the gene product corresponding to each tag is given, followed by alternative names in parenthesis.

no.	Tag Sequence	N-EC's	T-EC's	HUVEC	HIMVEC	Cell Lines	Description
1	TCTCAGGTCT	28	0	0	0	0	mucosal vascular addressin cell adhesion molecule 1
2	GTAGCGTTT	19	0	4	14	0	serum deprivation response (phosphatidylinositol-binding protein)
3	GTGGCTGACG	18	0	1	0	0	ESTs / intercellular adhesion molecule 4
4	CTCTTAAAA	34	1	1	0	0	small inducible cytokine subfamily A (Cys-Cys), member 14
5	TGGGAAGAGG	16	0	3	4	1	ESTs
6	GTTTAAGGAT	16	0	0	0	0	ESTs
7	CTTTGTTTG	15	0	56	32	1	endothelin 1 / ribosomal protein L27
8	ATTGCAATC	14	0	0	4	0	TU3A protein
9	TGTTGAAAA	21	1	1	0	0	selectin E (endothelial adhesion molecule 1)
10	ACAAAAAGC	21	1	0	6	0	TU3A protein
11	AAGATGCAC	21	1	1	1	1	phosphodiesterase 1 - nucleotide pyrophosphatase 2 (euloxin)
12	TAGAGGAAA	10	0	0	9	0	platelet/endothelial cell adhesion molecule (CD31 antigen)
13	TGTTTCAAG	10	0	0	1	0	ESTs
14	CTCTTCAAAA	19	1	1	0	0	ESTs / small inducible cytokine subfamily A, member 14
15	TATTAATAA	18	1	6	1	1	transforming growth factor, beta receptor II (70-60KD)
16	GAATTAACCA	9	0	1	14	0	ESTs
17	AAGGAGAACT	9	0	1	0	0	small inducible cytokine subfamily A, member 14
18	AATATCTGAC	9	0	2	2	2	active BCR-related gene
19	TCAGTGAACG	17	1	4	7	2	protein kinase C α
20	GCAAGTGCC	32	2	1	5	0	ESTs
21	TAAATCTTG	8	0	2	0	0	ESTs (2 unigene clusters)
22	GTCACTAAT	8	0	1	0	0	ESTs
23	ATAACCTGCA	8	0	0	0	0	signaling lymphocytic activation molecule
24	TGATCTGTGC	46	3	1	1	0	ESTs / glycogenin 2
25	TAAAGGCACA	15	1	4	3	0	LIM binding domain 2
26	GACCGCGGCT	73	5	11	7	0	claudin 5
27	ACTCGGGTGT	14	1	0	8	0	ESTs

		27	2	3	1	0	GTP-binding protein
28	CTTCTACCT	13	1	0	0	0	ESTs
29	TCGTGCTTTG	13	1	4	2	1	feline sarcoma viral (v-fes) - Fujinami avian sarcoma viral (v-fps) homolog
30	GAGCAGTGCT	10	1	0	1	0	ESTs
31	CTCTAAAAA	10	1	0	0	1	phospholipase C, beta 4
32	GAACCCCGT	10	1	7	15	1	ESTs
33	AACACAGTGC	10	1	7	15	1	ESTs